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The Journal of NERVOUS AND MENTAL DISEASE

An Educational Journal of Neuropsychiatry, founded in 1874

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Published monthly by

THE WILLIAMS & WILKINS COMPANY

BALTIMORE 2, MARYLAND

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The Journal of Nervous and Mental Disease

Vol. 121, No. 3 MARCH 1955 Serial No. 879

CONTENTS

Periventricular Diffuse Pinealoma. M. A. Neumann 193

Psychological Abnormalities in Agencies of the Corpus Callosum. J. R. Russell and R. M. Reitan 205

Latent Epilepsy as a Factor in Tension States. D. W. Liddell, A. R. Ashley and C. E. Darby 215

Over-Identification in the Schizophrenic Child and Its Relationship to Treatment. M. Owen 223

Psychosexual Identification in the Epilepsies. K. Bhaskaran 230

Convulsions in Childhood and Epilepsy. F. Sal y Rosas 236

Changes in Thematic Apperception Test Stories Paralleling Changes in Clinical Status of Schizophrenic Patients. R. Goldman and M. Greenblatt 243

Transference and the Physical Presence of the Physician. P. Lowinger and P. E. Huston 250

Studies in Alpha Blocking Following Electroconvulsive Therapy. A. N. Browne-Mayers and F. D. Kane 257

Intoxicant Drugs (Mescaline and Lysergic Acid Diethylamide) in Psychotherapy. W. Frederking 262

Sudden Death in Insulin Coma Treatment for Schizophrenia. A. Leitch 267

Characteristic Headache Resulting From Prolonged Use of Ergot Derivatives. C. W. Lippman 270

Persistent Sensory Symptoms Following Spontaneous Hypoglycemia Response to Chlorpromazine. W. H. Trethowan 274

Society Proceedings

New York Academy of Medicine: Section on Neurology and Psychiatry and the New York Neurological Society Meeting, January 11, 1955. 279

New York Neurological Society and the New York Academy of Medicine: Section on Neurology and Psychiatry Meeting, February 8, 1955. 282

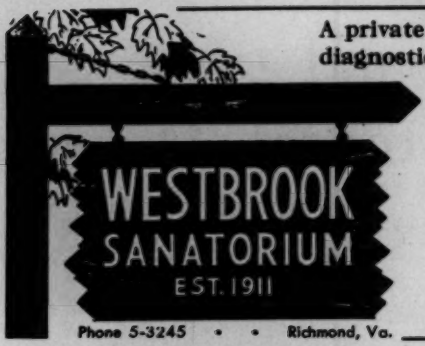
Boston Society of Psychiatry and Neurology Meeting, May 20, 1954. 284

Boston Society of Psychiatry and Neurology Meeting, November 18, 1954. 287

Boston Society of Psychiatry and Neurology Meeting, December 16, 1954. 289

Book Reviews 293

Notes and News 295



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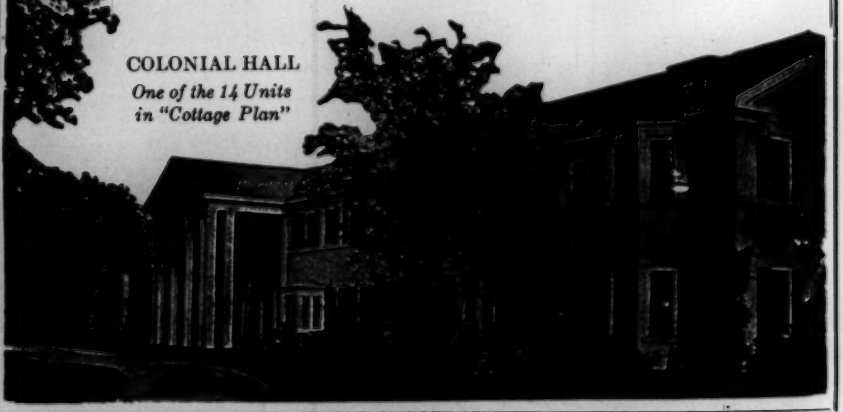

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Vol. 121, No. 3 MARCH 1955 Serial No. 879

PERIVENTRICULAR DIFFUSE PINEALOMA

Report of a Case with Clinical Features of Catatonic Schizophrenia

META A. NEWMANN, M.S.

Organic diseases of the central nervous system occurring in catatonic or schizophrenic (dementia precox) patients have been reported on several occasions (12, 13, 32, 17). In some of these instances the structural changes in the brain have been considered responsible for the deviation from normal behavior exhibited by the patients.

The patient whose history is presented in this study was, throughout his relatively brief illness (3 months' duration), consistently diagnosed as a typical case of catatonic dementia precox. The neurologic findings, although indicative of disturbance of hypothalamic function in retrospect, were believed to be of psychogenic origin. The pathologic changes were confined almost entirely to the regions surrounding the third ventricle (thalamus, hypothalamus and pretectal region).

Fulton (17) in his discussion of the clinical implications of the hypothalamus calls attention to the fact that psychiatric alterations are frequent accompaniments of lesions in this area (18, 21, 1, 2). From the work of various investigators it has been concluded that lesions of the anterior hypothalamus tend to produce excited states, while posterior lesions are likely to be associated with lethargy, indifference and depression with a tendency toward catatonia (18, 21). Alpers (1) states: "In general, diseases of the hypothalamus in man produce the following clinical features: 1) emotional changes; 2) intellectual deficits; 3) personality disorder; 4) psychotic manifestations."

In view of Fulton's belief that more careful correlations should be made between organic lesions of the nervous system and mental aberrations, the case is presented below in detail.

Case History

A 23-year-old white male was admitted to St. Elizabeths Hospital on May 3, 1945, after 2 years and 7 months of military service.

The family history is meager, but as far as known no mental disease existed. The patient's mother died of unknown cause when he was about two years old. She had three other children by a previous marriage. The patient's older sister was married, well, and furnished apparently reliable data. When the patient was 5 years old he was sent to an orphanage following a charge of neglect against the illiterate father. Shortly thereafter he was placed in a foster home and remained out of contact with his relatives until a few years prior to his enlistment in the service.

In his childhood he was described as quiet and well-behaved. He did poorly in school at first, but later obtained average grades. He stole money from his foster parents on at least two occasions. When he was 12 years old, he was physically immature, highly emotional and cried easily. At the age of 15 years instances of truancy were reported and he was transferred to another foster home. In January 1938 he began to fail in his studies and, according to the foster mother, "acted peculiarly, was very quiet, and went to bed early of his own volition." After he completed the tenth grade of school, at the age of 17 or 18, he worked at various odd jobs prior to his enlistment in the Navy.

About this time he resumed contact with his siblings. The sister stated that the patient was "difficult to understand and she always felt there was something on his mind." Even while visiting his relatives he usually remained by himself. "He would sit in a corner and just stare into space."

The only recorded illnesses were diphtheria at the age of 5 years, and bronchopneumonia. He did not use alcohol or drugs.

While on duty in the Canal Zone, the patient de-

veloped odd mannerisms and involuntary urinary habits. These "peculiar" actions manifested themselves during a visual acuity test and led to hospitalization on March 1, 1945. Previous visual tests (August 21, 1944, September 1944 and February 15, 1945) had shown a left hyperphoria. Although he had been noted to be mildly seclusive, this trait now became exaggerated and was accompanied by bizarre posturing. The strange positions were maintained for long periods of time. He wet himself either when standing or in bed, at about one-hour intervals. He seemed unconcerned about this, stating he had done so from childhood, but his shipmates claimed they had not noticed this previously. He spent most of his time in bed, sleeping or lying with his eyes closed, speaking to no one. When he did get up, he walked "as if in a dream, ate very little and appeared to be living in a world of his own."

History was obtained from the patient while he was in this state of retardation. It was observed that while he had difficulty in concentrating he appeared to be trying to give the correct replies. He replied to questions chiefly in monosyllables and was partially disoriented. The facies were expressionless, and he showed no interest in his surroundings. Although he appeared to be acutely ill the physical examination was essentially negative. The pupils were round and equal and reacted to light and in accommodation. The diagnosis after this examination was constitutional psychopathic state, schizoid personality.

He was transferred to a Naval Hospital April 17, 1945. The notes stated: "The patient's behavior since he has been in this hospital has been typical of a catatonic schizophrenia."

On admission to Saint Elizabeths Hospital the patient stated he had been all right until about three weeks previously when his legs "went out, then my jaws." When asked why, he replied, "Probably a cold" but when questioned if he had a cold, he replied in the negative. When asked if anything was wrong with him he answered, "Just my legs and weak kidneys, can't hold water." He said that nothing was wrong with his mind, and in an indifferent manner denied delusions of reference, hallucinations, worries, depression or suicide. His gait was unsteady, shuffling and short-stepped. He was extremely insecure in all movements. He sat quietly, staring off into the distance. He offered no spontaneous speech, and appeared in no pain or distress.

Examination (May 3, 1945) showed dilated pupils, fixed to light with a questionable slight hippus reaction; extraocular movements were apparently normal. The disks seemed pale but the edges were distinct. The deep tendon and superficial reflexes were equal throughout but very brisk; there was poorly sustained ankle clonus bilaterally. On passive movements, the muscle tone was decidedly diminished. There was swaying in the Romberg position, with eyes both open and closed. He was unable to perform any tests requiring co-ordination. The tongue showed marked fine and coarse tremors. A roentgenogram of the head, May 11, 1945, was reported negative for a tumor in the region of the sella turcica.

The notes stated that since admission the patient's condition had shown considerable variation, in both clinical, psychiatric and neurological symptoms. At times he was in good contact and was able to walk, while on other occasions he was semi-stuporous. He continued incontinent of urine and feces. Although he drank copious amounts of fluids, he still appeared dehydrated (May 7). His speech was scanning, slurred and inarticulate. He fell on several occasions: once from a wheel chair (May 8) and shortly afterwards while walking unassisted. On this latter occasion he became rigid and fell backward, but did not lose consciousness. When he was picked up from the floor he was still quite tense. On May 13 while walking to the cafeteria he slumped forward to the floor, making no effort to break his fall. On May 12 it was necessary to tube feed him. On this date the notes state that he took water and other liquids sparingly, and that he had lost considerable weight.

The picture—both mental and physical—was one of extreme lethargy, apathy and weariness. Under sodium amytal narcosis there was exaggeration of lethargy and persistence of pathologic reflexes. The consensus of opinion was that the patient's condition was essentially due to catatonia.

On May 15 he seemed weak and listless and complained of vague pains in his legs. His temperature rose to 103.8° during the night. He asked for water constantly and voided large quantities frequently. A spinal tap (May 17) showed no elevation of pressure. Slight nuchal rigidity was reported on that date. He received sulfamerazine and intravenous injections of glucose. When his temperature rose to 105° on May 19, penicillin therapy was instituted. After a slight chill of 15 minutes' duration during the night of May 20, the temperature dropped to 103°.

He received water in large quantities in response to his requests, almost every hour during the night of May 21. Polyuria continued. His blood pressure that day was reported at 85/50; pulse rate was 70 and feeble. He had difficulty focusing his eyes, but extra-ocular movements were intact. On May 25, there were signs of pulmonary pathology. He did not improve with sulfonamide or penicillin therapy. He became comatose, cyanotic and unable to swallow. The temperature rose to 108 at 2 a.m. on May 27, and a few hours later he ceased to breathe. The impression of the clinician was that death was due to aspiration pneumonia.

Throughout hospitalization the temperature had fluctuated. It was recorded as above 100° between May 16 and 21; about noon that day it dropped to just a little above normal, rose in the afternoon to 104.5°; dropped to slightly below normal on the morning of May 22. It remained subnormal till the morning of May 25, then rose steadily through May 26 until death.

Laboratory data. Hematologic, serologic and bacteriologic studies were noncontributory.

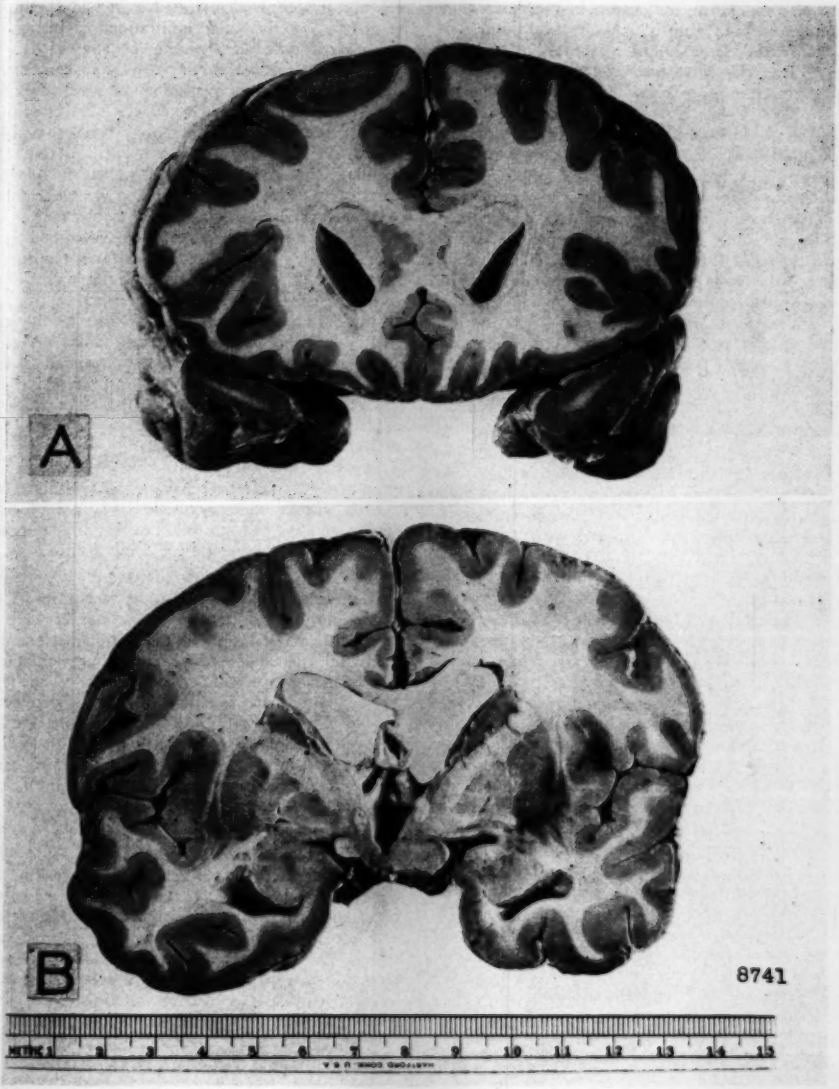

Fig. 1. A, sharply demarcated lesion of corpus callosum. *B*, tumor in right optic tract and tuber cinereum.

Necropsy findings[1]

The cause of death was given as pulmonary infarction. It was specifically stated that no arterial block was demonstrable. The pulmonary vessels showed a healthy condition. No exudates were recognized but a moderate degree of hypostasis was present. The heart was somewhat dilated. The aorta and prostate were hypoplastic. The testes were small, but were described as healthy on cross sections. The small brain (weight 1070 grams) was edematous. It was noted that the floor of the third ventricle bulged somewhat. Cranial nerves appeared normal.

Microscopic examination (visceral organs). The lungs showed an early bronchopneumonia. There was

[1] Autopsy was performed 30 hours after death by Dr. K. H. Langenstrass.

a trace of lymphocytic infiltration in the epicardium and minute areas of fibrosis in the myocardium. The liver, pancreas, adrenal gland and prostate appeared normal. The kidney showed congestion and stasis. Surprisingly, the testes showed widespread fibrosis and atrophy. The basement membranes of the tubules were thickened. Sertoli cells in some instances filled the lumen completely. A trace of spermatogenesis was indicated by a rare mitotic figure, but no mature spermatocytes were observed. Occasional plasma cells were noted in the dense interstitial tissue. The spleen showed hyperemia, stasis and diffuse hemosiderin pigmentation. The thymus, which grossly was described as replaced by fibroadipose tissue, was quite cellular. Occasional plasma cells were scattered in the parenchyma. No normal Hassal bodies were observed: they were either completely or almost completely hyalinized. The pituitary gland showed abundant eosinophilic cells in the anterior lobe. Lymphocytes and plasma cells were observed in the pars nervosa.

Cerebral pathology (gross). After fixation the brain was sectioned coronally. Several lesions were described: in the genu of the corpus callosum; right lateral wall of third ventricle at the anterior level of the hypothalamus, right optic tract and infundibulum; bilateral periventricular regions of the thalamus; and pulvinar. The condition was diagnosed "ependymomatosis." The ependyma was everywhere smooth. The pineal gland was believed involved in the neoplastic process.

Several months later the brain was reexamined. The lesion in the genu of the corpus callosum had a grayish gelatinous appearance and was sharply demarcated from the surrounding white matter. It thus resembled a demyelinating process rather than a neoplastic one (fig. 1 *A*). In a section through the descending columns of the fornix and amygdala, the right ventral hypothalamic area and optic tract was swollen, soft and faintly grayish (fig. 1 *B*). The lesion was separated from the more dorsal regions of the hypothalamus by a thin pinkish zone. In the section at the level of the corpora Luysii the bilateral thalamic lesions were faintly outlined; they caused no compression of adjacent thalamic structures. The centrally placed pretectal lesion was also faintly demarcated and caused no increase in volume. The pineal gland was only slightly enlarged. No other focal lesions were observed on detailed inspection. Against the definitive diagnosis of neoplastic disease was the absence of cerebral compression, and presence of swelling only in the region of the right optic tract. The short duration of the patient's illness and the questionable neurologic signs led to the impression that this was a case of acute multiple sclerosis, although only the callosal lesion resembled the plaques observed in demyelinating diseases.

Numerous sections for histologic study were selected and stained by a variety of general and specific histologic methods.

Microscopic examination. Preliminary study of the corpus callosum section showed extensive loss of myelin sheaths and axis cylinders in the lesion, which was not as sharply demarcated as it had appeared to be grossly. Perivascular infiltrations of lymphocytes and plasma cells were prominent (fig. 2 *A*). Between the vessels were many closely packed vesicular nuclei showing some variation in size and shape. Some cells were multinucleated, the number of nuclei seldom exceeding three. No processes could be demonstrated on these cells by specific stains. Fine granules, observed in some cells, may be blepharoplasts; these were not recognized in other lesions. Occasional mitotic figures were observed. Gemästete glia and macrophages were identified at the edge of the lesion where the cellular reaction was less dense. A connective tissue framework throughout the demyelinated area diminished at the edge of the lesion where it was confined to the vessel walls, enclosing the small dark cells within the reticulum strands. In the well myelinated portion of the corpus callosum, there were several discrete foci composed of a mixture of the large paler cells and small dark ones. Sometimes a small vessel was observed within these foci. Rarely small gland-like spaces lined with cuboidal cells were noted. The prominence of plasma cells and perivascular cuffing in this section was suggestive of a granulomatous disease process but no acid-fast bacilli, spirochetes, or other organisms could be demonstrated.

In the right hypothalamus there was complete demyelination of the optic tract except in its most lateral portion, while all other structures were well myelinated. The lesion in the optic tract and tuber cinereum was composed of an intimate mixture of the types of cells described above. The typical mosaic pattern and the fibrous stroma were indicative of a neoplasm of pineal origin (fig. 2 *B*). A few multinucleated giant cells were observed, and occasional mitotic figures. Perdrau stain demonstrated only occasional strongly argyrophilic fibers in the abundant stroma.

An almost symmetrical section from the left hypothalamus showed extension of the right-sided lesion in the floor of the third ventricle. In addition there were several minute foci composed of the two types of cells scattered in the wall of the third ventricle but separated from the ependyma by normal parenchyma. A focus of granulocytes and large oval or kidney-shaped pale nuclei was observed in the center of the optic tract. There was little if any fibrous stroma in this lesion. Several mitotic figures were observed peripherally. Another focus with the characteristic mixture of the two types of cells in a connective tissue framework, but with the addition of polynorphonuclear leukocytes, was at the junction of choroid roof of the third ventricle with the taenia thalami. In this same section, lateral to the descending column of the fornix, was an isolated giant cell surrounded by a few small dark cells. There were perivascular accumulations of lymphocytes and plasma cells. In one instance, at least, a mitotic figure was observed among these cells, and other mitoses were scattered in the parenchyma near the wall of the third ventricle, dorsal to the hypothalamic sulcus.

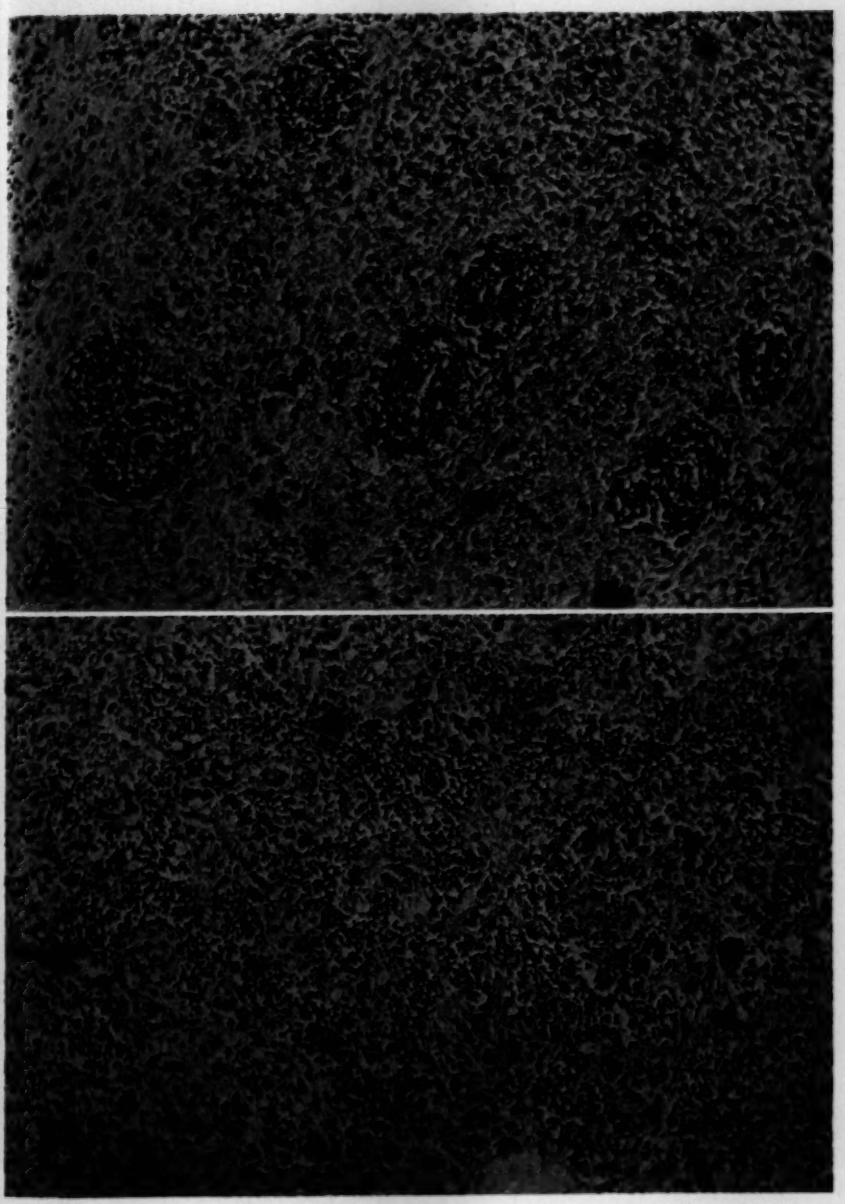

Fig. 2. A, growth in corpus callosum, showing perivascular arrangement of small dark cells; cresyl violet stain, 16 mm. objective. *B,* typical mosaic pattern of pinealoma in right optic tract region. Note multinucleated giant cell; hematoxylin-eosin, 16 mm. objective.

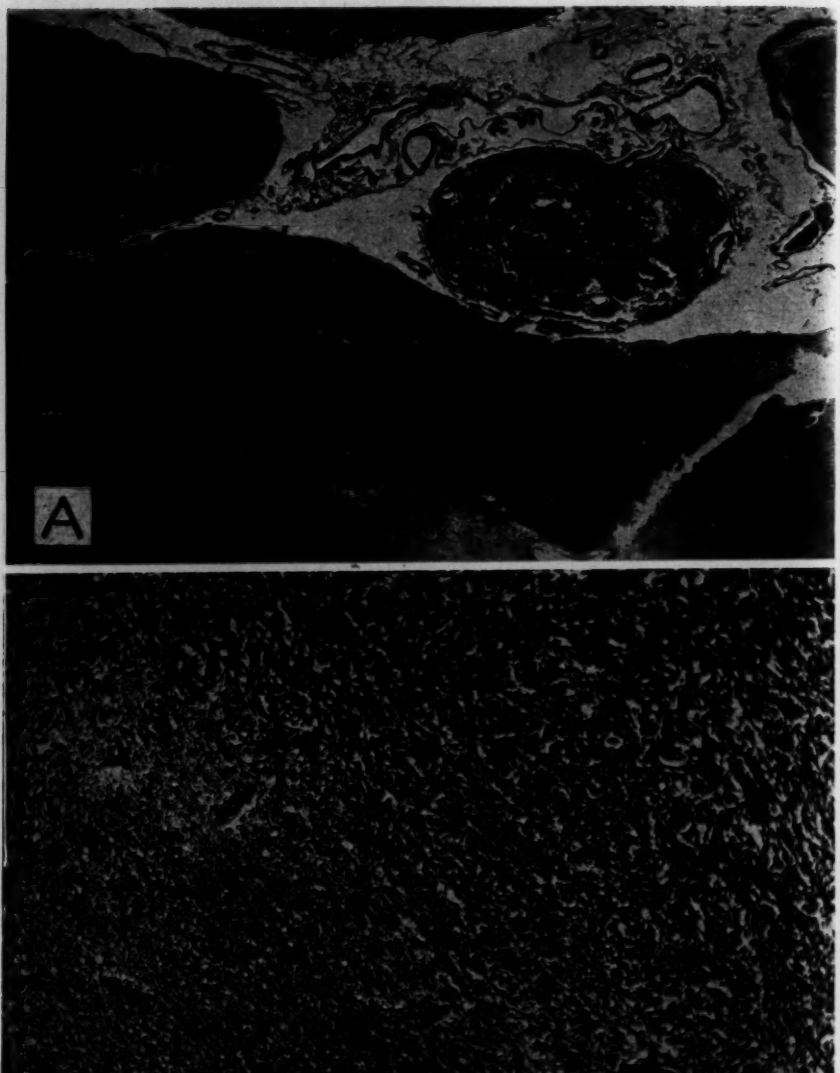

Fig. 3. A, neoplasm in pretectal region and pineal gland; phosphotungstic acid hematoxylin stain, 64 mm. Summar. *B,* collagenous meshwork in pretectal growth; Heidenhain collagen method, 16 mm. objective.

The supraoptic nucleus was not identified at all in the right hypothalamic section, since it was replaced by tumor cells, although ordinarily it should be prominent at the level studied. On the left only a few pale disintegrating nerve cells remained of the nucleus.

In a section through the posterior hypothalamus, the characteristic lesion was found along the wall of the third ventricle, dorsal to the hypothalamic sulcus and spreading into the medial thalamic nucleus. Pattern formation was most prominent in the lateral portion of the periventricular growth. In other areas there were almost concentric arrangements of the large pale cells surrounded by small dark nuclei. Giant cells were infrequently observed. Mitotic figures were noted not only in the main neoplastic process, but also in small isolated foci in the adjacent thalamus. In addition there were isolated mitoses in cells relatively distant from tumor formation. Pale swollen nerve cells with eccentric nuclei and in stages of dissolution were found in areas close to the growth and in the mammillary body, and caudate nucleus. In the lateral thalamic nucleus and posterior hypothalamus almost all of the nerve cells showed ischemic homogeneous changes.

The right caudal visual pathway also showed marked loss of myelin sheaths and axis cylinders. The remaining sheaths were fragmented and beaded; there were also myelin "balls." Connective tissue was hyperplastic only in the perivascular areas. There was reactive gliosis; the astrocytes were hypertrophied.

In the periventricular lesion in the left thalamus, the midline nuclei were almost completely destroyed. The neural parenchyma was replaced by the characteristic conglomeration of cells in irregular pattern lying in an argyrophilic reticulum. A few of the tumor cells showed tail-like processes, thus resembling spongioblasts. The nucleolus was prominent in many ot the large cells. Gemästete glia were numerous within the neoplastic process; while peripherally there was moderate cellular and fibrillary gliosis. Transitional forms from microglia to gitter cells were observed at the borders of the cellular focus. Mitotic figures were abundant in this region, and were occasionally noted in the walls of capillaries, as well as in isolated cells distant from the lesion. Some of the degenerating nerve cells adjacent to the growth had bizarrely shaped pyknotic nuclei.

Section through the pulvinar, epiphysis and pretectal region included a centrally located lesion just beneath the pineal gland (fig. 3 *A*). In the center of the epiphysis was a cluster of calcified nodules. The dense fibrous stroma was irregularly distributed; in its meshes were clusters of rounded cells with large vesicular nuclei interspersed with lymphocytes and plasma cells. The various cell types formed a less definite pattern than in the cerebral foci. Giant cells with a rim of small nuclei (thus resembling Langhans cells) were observed in several preparations. Some of the smaller pale-nucleated cells resembled the cell type found in normal pineal glands, and lacked a prominent nucleolus. The growth of cells in the pretectal region appeared much more uncontrolled than in the pineal gland. For example the large cells of the epiphysis were fairly uniform in size and shape, while in the pretectal lesion they showed considerable variation in size and shape, and multinucleated cells were more numerous, particularly binucleated ones. In some parts of the lesion the large cells were practically absent; here the small dark cells populated a dense fibrous stroma. The entire midportion of the tectum was destroyed, but not in the periagueductal region. On one side the process extended into the ventral portion of the pulvinar. The lesion was surrounded by a zone of cellular and fibrillary gliosis. In the phosphotungstic acid hematoxylin and Heidenhain preparations, the glial fibrils were intimately mixed with the heavy collagenous fibers of the stroma (fig. 3 *B*).

Grossly the mesencephalon had appeared normal. Surprisingly, widespread changes were observed, manifested by alterations in the glia and vascular endothelium. In the tectum numerous mitotic figures were noted (fig. 4 *A*). These were generally in isolated cells. Mitoses were also observed in a small focus of large pale cells just ventral to the colliculus on one side, slightly below the level of the aqueduct (fig. 4 *B*). The nuclei were round, oval or faintly notched; there was scanty chromatin. Cell body outlines were indistinct. There were no glial fibrils in this focus. Astrocytes throughout the section were generally swollen and hypertrophied or of Nissl's plump type. In some the expansions were fragmented, exhibiting the clasmatodendrosis of Cajal. Large pale swollen nuclei, with no demonstrable perikaryon and no processes, could not be definitely classified: they were believed to be macroglia in a more advanced stage of degeneration. They were generally noted to lie in areas showing damaged myelin sheaths. Weil stain demonstrated myelin loss in one colliculus, and more ventrally, on the same side, in a region lateral to the oculomotor nucleus. The tecto-spinal fibers appeared intact but fibers coursing in the other directions and mingling with the tecto-spinal bundles showed beading and fragmentation. On the dorso-lateral curvature of each colliculus there was a thick layer of glial fibrils. Since some of the numererous mitoses were actually seen in this glial layer, the question of the type of cell in which this sign of proliferation was occurring is of interest, since astrocytes are said to multiply by *amitotic* division only. Binucleated cells, possibly instances of amitotic division were also observed. Hortega cells were not prominent.

The periaqueductal region showed evidence of neoplastic activity. In one section a small mixed focus of large pale cells together with a few plasma cells and lymphocytes was observed in the floor of the aqueduct in its central depression and appeared to have broken through the ependymal lining. A scanty collagenous meshwork was found in this focus. In another section there were several small clusters of neoplastic cells; one focus included a multinucleated giant cell (fig. 4 *C*); a few such

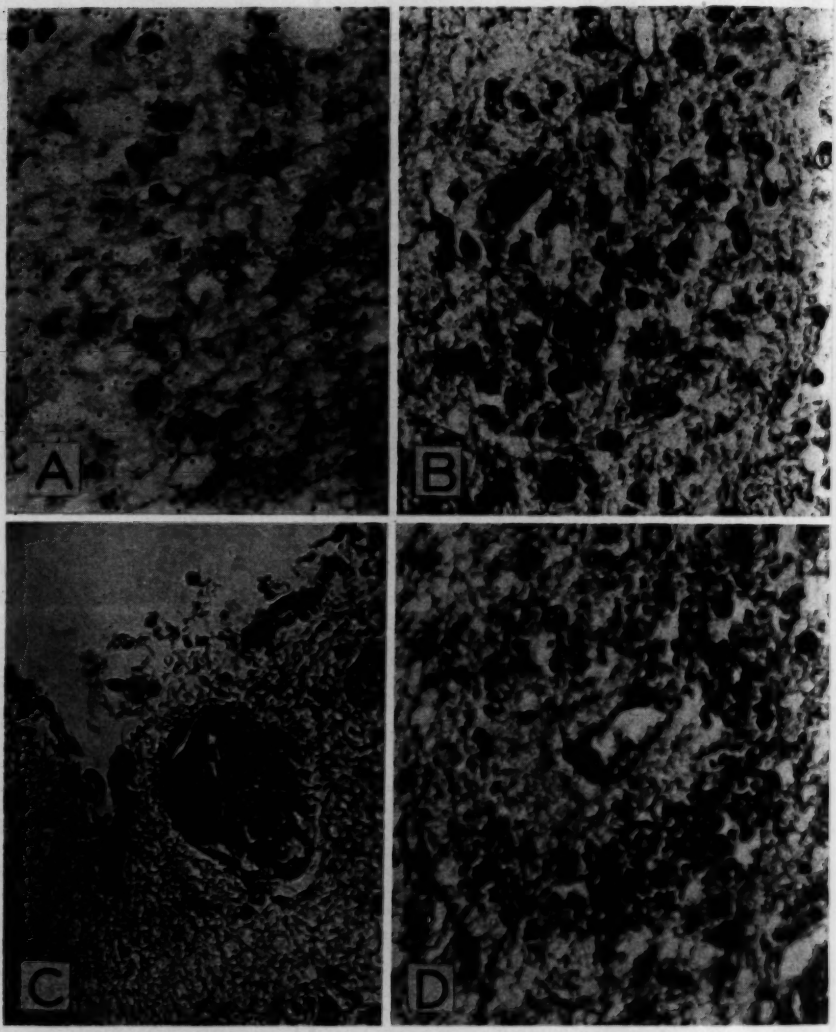

Fig. 4. A, three mitotic figures in tectum. *B,* focus of neoplastic cells in mesencephalon, with mitoses. *C,* neoplastic focus with giant cell close to aqueduct. *D,* mitosis in capillary wall; ring hemorrhage can be faintly seen. Hematoxylin-eosin stain, 4 mm. objective.

cell groups had mitotic figures in their midst, and in one group low cuboidal cells were arranged around a small cavity, thus suggesting the architecture of an ependymoma. Some of the scattered cell groups were separated from the ependyma by a zone of fibrillary gliosis, others appeared to spread from the apparently normal cuboidal cells lining the aqueduct. The cells in these groups were less differentiated than those in the larger periventricular lesions.

In the area of myelin loss in the tegmentum described previously, two minute recent hemorrhages were observed. One of these petechiae was around a small vessel but was separated from it by a narrow zone of necrosis. In the vessel wall were two enormously swollen endothelial cells and one mitotic figure (fig. 4 *D*). All vessels in the area were lined with swollen cells, and scattered throughout the area were other swollen cells with pale nuclei (gitter cells?). In a Weil preparation a focal area of pallor was noted in one oculomotor nerve as its

fibers were passing into the interpeduncular fossa. Between the thickened and widely separated axis cylinders were scattered macrophages.

In the left frontal polar section myelin sheaths were thinned out in the white matter where rare minute foci of large pale cells intermingled with small dark ones were observed. There was no connective tissue proliferation in these foci, except around small blood vessels. A few degenerating astrocytes were scattered in the thinned-out subcortex. In this section and in other cortical areas, nerve cells showed mild non-specific changes: some were undergoing lysis, and many had pyknotic nuclei. Lymphocytes were rarely observed in perivascular spaces or near ependymal granulations. A mitotic figure was observed in a small focus of mixed cells just beneath the ventricular surface of the inferior horn. The slightly thickened leptomeninges of all cortical sections showed mild focal infiltration with lymphocytes and plasma cells.

In the cerebellum and lower brainstem sections acute nerve cell changes were fairly prominent. There was focal proliferation of glia in Bergmann's layer, and moderate proliferation of microglia in the inferior olives. No neoplastic foci were observed. Congested vessels and a few petechiae were noted, the latter generally in the gray matter. There were very rare perivascular infiltrations of lymphocytes and macrophages; these were never large.

Comment and Review of the Literature

The periventricular lesions described in this presentation were clearly neoplastic in nature. The structure of the growth was characteristically that of a pinealoma. The histologic structure of this type of tumor has been described by many observers (22, 3, 20, 33). One of the most thorough studies was that of Globus and Silbert (20), in which the varying histologic picture observed is explained by the fact that the normal gland passes through several distinct histogenetic stages, any one of which may be duplicated by the tumor growth. The widespread distribution of the growth around ventricular spaces with little or no involvement of the pineal gland is rare but not unique. Ford and Muncie (15) reported three such cases, Mackay (25) and Riggs (31) each one. The picture in the reported case is similar to these. In none was the pineal gland greatly enlarged; in fact in Riggs' patient the pineal gland was not even found. Mackay believed that the neoplasm did not originate in the gland but invaded it secondarily. His hypothesis that the growth had a diffuse ependymal origin was strengthened by the fact that definite ependymal structures and cell forms were scattered throughout the tumor, which histologically had the structure of a pinealoma. This was also noted in the reported case. Since the pineal gland develops as an ependymal outbudding (10) it is not surprising to find ependymal-like structures within a pinealoma. Ford and Muncie (15) considered the possibility that their reported tumors sprang from embryonic remnants, as they state that "a number of vestigial structures are observed in the walls of the third ventricle during embryonic life. Although these disappear long before birth they may leave cell rests which are capable of giving rise to a tumor." Bailey (3) postulated that such tumors may have arisen from other primordia near the pineal anlage.

A feature that has heretofore not been stressed is the apparent spread not only by implantation on ependymal surfaces by means of the cerebrospinal fluid, or perhaps a multicentric origin from minute cells rests in these areas, but the suspected infiltration of the brain either by the blood stream or following the course of capillaries. This is evidenced by the scattered minute foci not in immediate proximity to the ventricles; a small vessel is nearly always observed in the centers. The presence of polymorphonuclear neutrophilic leukocytes in these presumably recent foci of new growth was at first puzzling, but is interpreted as a normal acute inflammatory reaction to a noxious agent —in this instance tumor cells. One or more mitotic figures were always present in these mixed cell groups. Just what significance should be attached to the endothelial mitoses in the capillary walls is not clear: it is possible they played a role in the formation of the hyperplastic reticulum noted in neoplastic areas, since it is known that endothelial cells may transform into fibroblasts (26).

The widespread occurrence of isolated mitotic figures in the parenchyma appears unique; it is not referred to in the literature of pinealomas. That they are a response of the microglia to an acute irritative process (40) is the most likely possibility. Mitoses in microglia are described generally as occurring in acute infections and intoxications (39). In my own experience, however, mitotic figures have been observed only rarely, and never as prominently as in the described case. Previously I have noted mitotic figures in the tectum in a few cases of

acute fatal catatonia. This evidence of active proliferation was indeed quite striking in the tectum of this patient, where there was no tumor involvement. Just what was the etiology of the slight loss of myelin and the degenerative changes in the remaining sheaths of the colliculi remains obscure. The degenerative changes in the astrocytes of this region, swelling, hypertrophy, and clasmatodendrosis of Cajal, are probably due to the same unknown influence. The fact that mitotic figures were also observed in the thick layer of glial fibrils on the dorso-lateral curvature of the colliculi and just ventral to this band, where astrocytes were hyperplastic, suggests that perhaps the mitoses were occurring in these cells, although Penfield (29) states that he has never seen mitotic division of astrocytes.

Several observers have pointed out the similarity of pineal tumors to seminomas (16, 38, 9). The duality of the cell types has also led to the suggestion that these growths should be included among the teratoid neoplasms (33, 38, 27, 34, 11). Both teratomas and pineal growths frequently show seeding in ventricles or subarachnoid space. The presence of discrete foci which appear to arise in the walls of, or around small vessels, is perhaps additional evidence of the participation of more than one type of tissue in this neoplasm. Russell and Sachs (33) stated that only two other types of tumor represent autochthonous teratoid growths, namely choriomas and thymomas. To this list should be added the hepatomas, which also reproduce the structure of hepatic parenchyma in distant metastases (28).

The symptoms of a single massive pineal tumor, usually in the region between the quadrigeminal plate and splenium of the corpus callosum, are quite different from the disseminated periventricular type of growth. These symptoms will not be discussed here since they are not pertinent to the study, and have been summarized by others (22, 20, 33, 30).

The patient's clinical symptoms can now be interpreted on the basis of the pathologic findings. The excessive thirst and polyuria are readily explained by the destruction of the supraoptic and paraventricular nuclei and also the degeneration of the suproptico-hypophyseal tract. There is no longer any doubt as to the role of these structures in the control of water metabolism. The existence of this center in the hypothalamus was conclusively demonstrated by the experiments of Gersh (19) and Fisher, Ingram and Ransom (14). The respective parts played by the pituitary gland and the hypothalamus in regulation of vegetative functions were, however, long controversial. The experiments of Bailey and Bremer (4, 5), conducted on dogs in 1921, established that minute discrete lesions of the parainfundibular area and tuber cinereum produced transient or permanent polyuria and polydipsia, depending on the size of the lesions. The integrity of the pituitary gland was maintained. Clinical investigations (18, 22, 33, 15, 31, 16, 38, 27, 36, 23) offered further convincing proof of the experimental observations.

The indecision regarding the endocrine nature of the pineal body has arisen from the fact that a certain number of children, suffering from tumors originating in that site, showed puberta precox, while in other children this symptom was absent. As Russell and Sachs (33) point out, the precocious development of adult sexual characteristics may result from hypothalamic involvement. No anatomic pathway, however, has yet been worked out for the control of sexual function or development, but transection of the stalk of the pituitary commonly caused disturbances of sexual activity in experimental animals and hypophysectomy caused atrophy of sex organs (26). The precise mechanism of the release of gonadotropic hormones of the pituitary gland into the blood stream has not been determined: One proposed mechanism is that it is on a neurovascular basis. The innervation of this gland has been demonstrated to originate chiefly in the supraoptic and paraventricular nuclei. Clinical confirmation comes from the reported instances of genital atrophy in cases of interruption of the hypothalamic-hypophyseal tract. Although the adiposo-genital atrophy is more common, there are reported cases of genital atrophy occurring without concomitant adiposity (8). Bailey and Bremer (4, 5) were able to produce cachexia and genital atrophy in their dogs as a result of moderately large lesions of the tuber cinereum. In this connection it may therefore be significant that the young man in the reported case had atrophic testes. Gonadal atrophy is seldom reported as a concomitant of pinealomas.

When the patient first came to the attention of the physicians it was stated that the pupils were round and equal and reacted to light and in accomodation. Several months later, when he was examined at Saint Elizabeths Hospital, the pupils had become dilated and fixed to light. This was apparently a constant finding, and can now be attributed to the lesion in the pretectal area.

The role of the hypothalamic lesions in causing the behavioral changes described in the patient cannot be definitely determined since the evidence is not conclusive. If the statements of the sister, who saw the patient only rarely, are accepted, he showed behavior suggestive of schizophrenic tendencies before he joined the service. Even experienced psychiatrists were unable to differentiate the patient's reaction pattern from true catatonia. Thus whether the "catatonia" was the result of long-standing hypothalamic involvement of a sleep center, or merely an exaggeration of early schizoid tendencies, must remain an unresolved question.

As noted in the introductory paragraphs, by no means all of the lesions reported in cases of schizophrenia have been in the hypothalamus. But the tendency of lesions in the floor of the third ventricle to produce somnolence, or profound lethargy closely simulating catatonia, is well known. Other forms of psychotic reactions have been reported concomitant with brain tumors (6, 35). Brock and Wiesel (7) discussed four patients in whom brain tumors were diagnosed only after a long period of psychotic manifestations. They stated "In the main, it would seem that these psychotic clinical pictures represent the reaction of an unstable personality to the presence of an organic disease, although one cannot be sure that the organic cerebral process may not sometimes induce some of the symptoms by direct effect on 'psychosomatic' mechanisms situated in the diencephalic-hypothalamic region."

An extensive literature discussing the relationship of the hypothalamus to hyperthermia, diabetes insipidus, disturbance of sleep mechanism and genital changes has developed (37). When these changes have occurred in conjunction with tumors of pineal origin, the growths have involved the tuber cinereum and interrupted the supraoptico-hypophyseal connections. The majority of these cases have occurred in young males; seldom has a patient with such a lesion reached the age of thirty.

Summary

1. A young man with a typical clinical picture of schizophrenia for three months, showed in addition diabetes insipidus, profound lethargy, dilated and fixed pupils, transient neurologic signs and terminal hyperthermia.

2. A diffuse neoplastic process, with the histologic structure of a pinealoma, surrounded the region of the third ventricle chiefly, involving also the pretectal region, and pineal gland. There was another focus in the genu of the corpus callosum and minute disseminated foci in the parenchyma, believed to follow the course of capillaries.

3. Pertinent literature of the pinealomas, and the relationships of the hypothalamus to the clinical symptoms, are reviewed.

Acknowledgements

Thanks are due to Dr. Peers, Chief, section on Pathologic Anatomy, Experimental Biology and Medicine Institute, National Institutes of Health, Bethesda, Maryland, for confirming the impression that the periventricular lesion was a pinealoma.

The excellent photographs are the work of Mr. Rudolph Torovsky, chief photographer of Saint Elizabeths Hospital.

BIBLIOGRAPHY

(1) Alpers, B. J.: Personality and emotional disorders associated with hypothalamic lesions. *Res. Publ., A. Res. Nerv. & Ment. Dis. 20:* 725. Baltimore: The Williams & Wilkins Co., 1940.

(2) Alpers, B. J.: Relation of the hypothalamus to disorders of personality. Report of a case. *Arch. Neurol. & Psychiat., 38:* 291, 1947.

(3) Bailey, P.: Cellular types in primary tumors of the brain, *Cytology and Cellular Pathology of the Nervous System, 3:* 903 ed. by W. Penfield. New York: Paul B. Hoeber, Inc., 1932.

(4) Bailey, P., and Bremer, F.: Experimental diabetes insipidus and genital atrophy. *Endocrinology, 5:* 761, 1921.

(5) Bailey, P., and Bremer, F.: Experimental diabetes insipidus. *Arch. Int. Med., 28:* 773, 1921.

(6) Bailey, P., and Murray, H. A.: A case of pinealoma with symptoms suggestive of compulsion neurosis. *Arch. Neurol. & Psychiat., 19:* 932, 1928.

(7) Brock, S., and Wiesel, B.: Psychotic symptoms marking the onset in cases of brain tumor. *Clin. North America, 32:* 759. Philadelphia and London: W. B. Saunders Co., 1948.

(8) Brooks, C. M.: Relation of the hypothalamus to gonadotropic functions of the hypophysis. *Res. Publ., A. Res. Nerv. Ment. Dis., 20:* 539. Baltimore: The Williams & Wilkins Co., 1940.

(9) Cairns, H.: Cited by Walton (38).

(10) Del Rio-Hortega, P.: Pineal gland. *Cytology and Cellular Pathology of the Nervous System, 2:* 635, ed. by W. Penfield. New York: Paul B. Hoeber, Inc., 1932.

(11) Ehni, George: Pineal teratoma: report of a case. *J. Neurosurg., 3:* 86, 1946.

(12) Ferraro, A.: Pathological changes in the brain of a case clinically diagnosed dementia precox. *J. Neuropath. & Exper. Neurol., 2:* 84, 1943.

(13) Ferraro, A.: Histopathological findings in two cases clinically diagnosed dementia precox. *Am. J. Psychiat., 13:* 883, 1934.

(14) Fisher, Ingram and Ransom: Cited by Fulton (17).

(15) Ford, F., and Muncie, W.: Malignant tumor within the third ventricle. Three cases of unusual type with invasion of the ventricular walls. *Arch. Neurol & Psychiat., 39:* 82, 1938.

(16) Friedman, N. B.: Germinoma of the pineal, its identity with germinoma ("seminoma") of the testis. *Cancer Research, 7:* 363. Philadelphia: The Donner Foundation, Inc., Cancer Research Division, 1947.

(17) Fulton, J. F.: *Physiology of the Nervous System,* ed. 2. New York: Oxford University Press, 1943.

(18) Fulton, J. F., and Bailey, P.: Tumors in the region of the third ventricle: their diagnosis and relations to pathological sleep. J. NERV. & MENT. DIS., *69:* 1, 145, 261, 1929.

(19) Gersh, I.: Water metabolism: endocrine factors. *Res. Publ., A. Res. Nerv. & Ment. Dis., 20:* 436. Baltimore: The Williams & Wilkins Co., 1940.

(20) Globus, J. H., and Silbert, S.: Pinealomas. *Arch. Neurol. & Psychiat., 25:* 937, 1931.

(21) Harrison, F.: The hypothalamus and sleep. *Res. Publ., A. Res. Nerv. & Ment. Dis., 20:* 635. Baltimore: The Williams & Wilkins Co., 1940.

(22) Horrax, G., and Bailey, P.: Tumors of the pineal body. *Arch. Neurol. & Psychiat., 13:* 423, 1925.

(23) Horrax, G.: The role of pinealomas in the causation of diabetes insipidus. *Ann. Surg., 126:* 725, 1947.

(24) Horrax, G., and Bailey, P.: Pineal Pathology. *Arch. Neurol. & Psychiat., 19:* 394, 1928.

(25) Mackay, R. P.: Pinealoma of diffuse ependymal origin. *Arch. Neurol. & Psychiat., 42:* 892, 1939.

(26) Maximow, A. A., and Bloom, W. A.: *Textbook of Histology,* ed. 6. Philadelphia & London: W. B. Saunders & Co., 1952.

(27) McGovern, V. J.: Tumors of the epiphysis cerebri. *J. Path. & Bact., 61:* 1, 1949.

(28) Neumann, M. A.: Primary carcinoma of the liver with metastases to bone. Report of a case. *Am. J. Path. 20:* 895, 1944.

(29) Penfield, W.: Neuroglia, normal and pathological. *Cytology and Cellular Pathology of the Nervous System, 2:* 421, ed. by W. Penfield. New York: Paul B. Hoeber, Inc., 1932.

(30) Posner, M., and Horrax, G.: Eye signs in pineal tumors. *J. Neurosurg., 3:* 15, 1946.

(31) Riggs, Helena E.: *Diffuse Involvement of the Brain by Pinealoma without Primary Tumor Formation.* Presented at the meeting of the Association of American Neuropathologists, Atlantic City, N. J., June 1953.

(32) Roizin, L., Moriarity, J. D., and Weil, A. A.: Demyelinization of the central nervous system in acute schizophrenia. *Arch. Neurol. & Psychiat., 54:* 202, 1945.

(33) Russell, W. O., and Sachs, E.: Pinealoma. A clinicopathologic study of seven cases with a review of the literature. *Arch. Path., 35:* 869, 1943.

(34) Russell, D. S.: The pinealoma: its relationship to teratoma. *J. Path. & Bact., 56:* 145, 1944.

(35) Smyth, G. E., and Stern, K.: Tumors of the thalamus. A clinco-pathological study. *Brain, 61:* 339, 1938.

(36) Stringer, S. W.: Diabetes insipidus associated with pinealoma transplant in the tuber cinerum. *Yale J. Biol. Med., 6:* 375, 1933-34.

(37) The hypothalamus and central levels of autonomic function. *Res. Publ., Res. Nerv. & Ment. Dis., 20.:* Baltimore: The Williams & Wilkins Co., 1940.

(38) Walton, Kenneth: Teratomas of the pineal region and their relationship to pinealomas. *J. Path. & Bact., 61:* 11, 1949.

(39) Weil, A.: *Textbook of Neuropathology,* ed. 2. New York: Grune & Stratton, Inc., 1945.

(40) Wertham, F.: *The Brain as an Organ.* New York: The MacMillan Company, 1934.

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PSYCHOLOGICAL ABNORMALITIES IN AGENESIS OF THE CORPUS CALLOSUM[1]

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REVIEW OF LITERATURE

The significance of the corpus callosum with regard to mental functions has long been a subject of interest and speculation. Several conditions have provided clinical material for study, including tumors, and more recently, agenesis and surgical section of the corpus callosum.

Agenesis of corpus callosum

Investigators of the effects of agenesis of the corpus callosum have been impressed with the paucity of indications of mental impairment usually found in this condition. Bruce (10) believed " . . . that if the brain is otherwise well developed, absence of the corpus callosum does not necessarily produce any disturbance of motility, coordination, general or special sensibility, reflexes, speech or intelligence." Cameron and Nicholls (11) reported, "Such meagre evidence as we possess seems to indicate that the collosal fibers are of much more importance in maintaining and governing the finer co-ordinations of muscular movement in the limbs of the opposite sides than in regulating the higher functions of mentality." These earlier investigators were handicapped by the necessity of studying the patients' lives retrospectively, after the postmortem examinations. Since the report by Davidoff and Dyke (15) of the method for diagnosing agenesis of the corpus callosum during life by encephalography, several writers have had opportunities to examine these patients clinically and to administer psychological tests. Hyndman and Penfield (21) reported psychological findings on a 21-year-old man with partial absence of the corpus callosum, enlargement of the lateral ventricles, and comparative atrophy of the left hemisphere. This patient obtained a rating of average intelligence and was able to read and comprehend written material at about the ninth grade level. Personality study showed no frankly psychopathic findings, although the patient seemed somewhat deficient in self-confidence, drive, and ability to plan and make decisions independently. Goldensohn *et al.* (16) have described the psychological status of an 11-year-old girl with agenesis of the posterior portions of the corpus callosum associated with internal hydrocephalus especially involving the third and lateral ventricles. This patient was rated as low dull average in intelligence. The Rorschach test, which has been shown to be fairly sensitive to the effects of organic cerebral pathology, (1) " . . . suggested the presence of an irreversible pathological state of the brain" (16). The patient was also described as showing a deficiency in conceptual or abstract thinking. Gowan and Masten (17) have reported the case of a 19-month-old child with partial agenesis of the corpus callosum and a " . . . marked degree of mental enfeeblement." Baker and Graves (9) describe a 14-year-old boy whose brain at necropsy showed only the earliest stage of development of the corpus callosum, after the formation of the septum pellucidum. He had a verbal intelligence quotient of 69, and his rating on performance tests ranged from slightly below this point to nearly an average level. These writers conclude that the mentality of patients with agenesis of the corpus callosum varies from idiocy to mediocre levels, corresponding somewhat with the associated anomalies of the brain. Kirschbaum (28) believes that absence of the corpus callosum is symptomless; that the variety of clinical manifestations seen may be accounted for by the associated malformations of the brain and frequently of other organs.

The apparent conclusion to be drawn from these reports is that agenesis of the corpus callosum results in no mental impairment unless it is associated with other brain anomalies. This conclusion would seem to be premature

[1] Supported in part by a grant from the James Whitcomb Riley Memorial Association.

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in consideration of the testing instruments used. In no instance has study of a patient with agenesis of the corpus callosum been made with standardized tests which were developed and validated for use in measuring the effects of brain damage. The tests used were ones standardized for measuring psychological abilities in a "normal" rather than brain-damaged population. The comparative insensitvity of these tests for determining the effects of brain damage is becoming increasingly clear as additional patients are being studied (18—20). Commenting on the relative failure of such tests to reveal the psychological effects of brain damage, Brickner (20) has said recently, "It is now known that many, if not all, of the conventional psychometirc tests fail to reveal the deficits shown in other ways by patients after operation."

Tumors of the corpus collosum

Publications (22—27) dealing with mental functions of patients with tumors of the corpus callosum are quite definite in their claim of impairment with callosal lesions. The symptoms attributed to collosal lesions have included poverty of movement and thought, torpor, apathy, depression, loss of memory, hallucinations, disorientation, emotional dulling, transient periods of motor excitement, inability to concentrate, motor apraxia, and imperviousness to external stimuli. Alpers (24, 25) has attempted to describe a mental syndrome specific for collosal tumors, in which the cardinal symptom is an imperviousness to stimuli, particularly auditory stimuli. He believes from the patients he studied that this symptom is associated with involvement of the corpus callosum per se.

Cass and Reeves (12) indicate a difficulty in drawing conclusions with regard to the function of the corpus callosum from the study of patients with gross lesions of the brain, such as may easily be true in the case of tumors. These writers caution that the inevitable involvement of neighboring structures will be misleading in such attempts. The possible role of increased intracranial pressure in such lesions should also be noted.

Our review of the literature fails to reveal a single instance of the use of standarized measuring instruments appropriate for evaluating the psychological effects of brain damage in tumors of the corpus callosum. Conclusions have been reached by the procedure of gross impressionistic observation, a method noted for its dubious reliability. It would seem, therefore, that the nature of possible psychological changes with tumors of the corpus callosum, and inferences with regard to the function of the corpus callosum from these studies, are still open to question.

Surgical section of corpus callosum

The function of the corpus callosum has also been studied in patients with surgical section of these fibers. Conclusions reached concerning the relation of the corpus callosum to mental functions have been distinctly different from those based on patients with callosal tumors. Dandy (13) partially sectioned the corpus callosum in removal of pineal tumors and evacuation of cysts of the cavum septi pellucidi and cavum vergae. He was unable to observe any mental changes following even extensive section of the corpus callosum, and concludes, "This simple experiment at once disposes of the extravagant claims to function of the corpus callosum (13). The patients in whom Van Wagenen and Herren (29) did callosal sections in an attempt to limit the spread of epileptic discharges have been studied quite thoroughly with regard to a variety of possible symptoms arising from the callosal lesions (29). Akelaitis (31, 34, 36–38) reports negative findings with regard to asterognosis, personality changes, visual, auditory, or tactile agnosia, language functions, the hemiamblyopic visual field, and size, object, letter, and color discriminative ability in either homonymous field of vision. Bridgman and Smith (39) report no change post-operatively in binocular depth perception, ability to maintain and recover fusion in response to diplopia-producing stimuli, and monocular apparent movement vision. Akelaitis (32, 33, 38) and Van Wagenen and Herren (29) found that dyspraxia did not occur unless damage to one of the cerebral hemispheres was also present.

Rather slight and sometimes transient positive symptoms were found. Smith and Akelaitis (30) report that some temporary shifts in laterality and a unilateral disturbance in motility occur in some patients. Temporary confu-

sion of orientation occurs only if unilateral involvement of the posterior part of the right or left hemisphere is also present (35). Bridgman and Smith (39) found some loss in divergence and convergence necessary to maintain and recover single binocular vision, but point out that the deficit is mild and of questionable significance. Some studies were done using formal psychological testing methods. Smith (41) studied pre- and post-operative ability in maze and mirror-drawing learning problems together with bilateral transfer. He found slight but inconsistent disturbances in maze learning and in bilateral maze transfer post-operatively, but no loss on mirror drawing problems. Parsons (40) used several standardized psychological tests pre- and post-operatively. The only significant change found was some impairment of immediate memory, reflected primarily by the Digit Span test. Interestingly, the Digit Span test has been found ineffective in differentiating traumatic brain-injured persons without dysphasia and neurotic controls in other studies (7, 8). Parsons used no control patients in her study.

There is obvious disagreement between conclusions regarding the function of the corpus callosum when one compares the studies made on persons with callosal tumors and callosal surgical section. The preponderance of mental symptoms (including intellectual dulling, apraxia, etc.) noted with callosal tumors are not reported with callosal section. Since tumors are so likely to involve other structures as well as the corpus callosum, one must respect the results from the surgical sections. Nevertheless, in these studies as with the others on the psychological significance of the corpus callosum, standardized psychological tests of demonstrated validity in measuring the effects of organic cerebral damage have not been used.

Case History

The patient we wish to describe in this report is a 19-year-old white female who in childhood was noted to have obvious mental retardation. At age 11 the child was committed to a state institution for mentally retarded children, at which time she was noted to have unsteadiness in walking and dysarthria. Two weeks prior to the hospital admission these symptoms increased and the patient began having failing vision. The patient began to vomit two days before admission.

General physical examination was not remarkable. Neurological examination disclosed the patient to be lethargic and mentally dull. Speech was slurred. Bilateral papilledema was present. There was a slight ptosis of the right eyelid. There was weakness of conjugate deviation of the eyes to the left and upward, and weakness of convergence. The deep reflexes

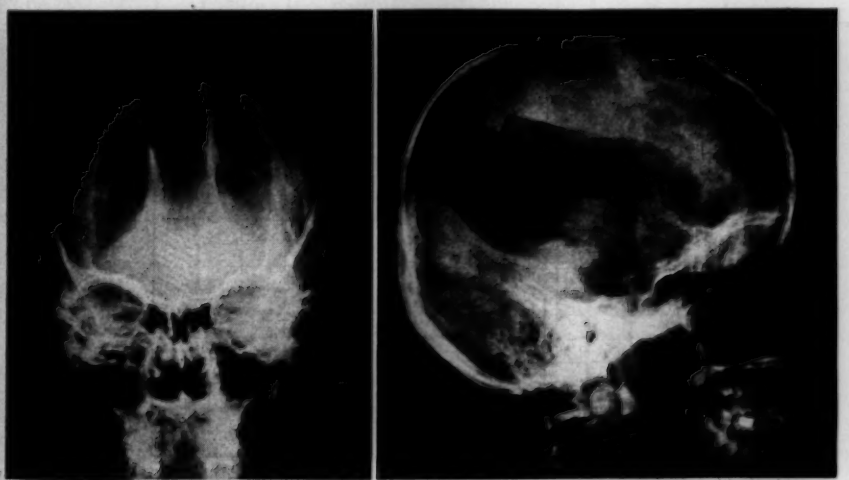

Fig. 1. Posterior-anterior and lateral views of ventriculogram of patient with agenesis of corpus callosum.

were depressed and the extremities were all hypotonic. There was bilateral ataxia of all four extremities and adiadokokinesis. The patient was unable to stand or walk.

Ventriculography revealed marked dilation of both lateral ventricles and the deformities characteristic of absence of the corpus callosum (fig. 1). The third ventricle was enlarged superiorly and posteriorly, the apex of the enlargement being just beneath the skull in the midline of the posterior parietal region. The aqueduct of Sylvius and fourth ventricle were not visualized by ventriculography.

Surgical exploration of the brain was carried out through a right occipitoparietal craniotomy and suboccipital craniectomy. This exploration revealed that the posterior portion of the falx was absent. Immediately beneath the superior sagittal sinus in the occipitoparietal region, and bulging between the cerebral hemispheres, was a thin membranous wall of the third ventricle. The foramina of Monro were widely dilated affording visualization of both lateral ventricles. The aqueduct of Sylvius could not be seen from above. However, dye placed in the third ventricle was recovered from the fourth ventricle, which was visualized by suboccipital craniectomy. The left cerebellar hemisphere appeared smaller than normal. Following this exploration the patient gradually improved. The papilledema disappeared. Vomiting ceased. Three months following the surgical exploration the patient showed impairment of left conjugate gaze. At that time she was able to walk without support, but was moderately unsteady. Some ataxia of the upper extremities remained but this was definitely less than preoperatively.

The greatest deformity in this patient was the complete absence of corpus callosum which, in conjunction with absence of the posterior portion of the falx, permitted the third ventricle to attain an unusual size and position. There was also maldevelopment of the cerebellum evident by gross inspection. The pathogenesis of the increased intracranial pressure and aggravation of neurological defects which brought the patient to the hospital is obscure. We have postulated that the patient may have had a temporary block in flow of cerebrospinal fluid through the aqueduct of Sylvius.

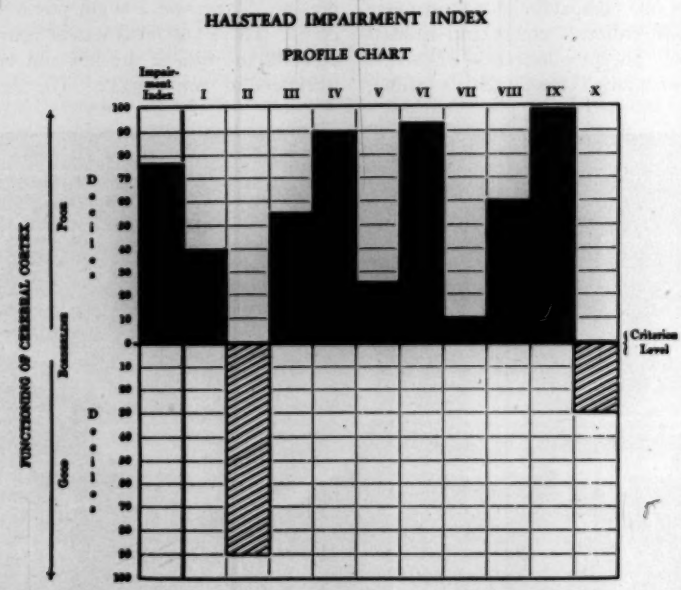

Fig. 2. Graphic presentation of mean values on Halstead's tests of biological intelligence obtained by patient with agenesis of the corpus callosum.

Psychological Studies of Patient

A large battery of psychological tests was administered to the patient three months following surgery. These tests included the neuropsychological indicators developed by Halstead (18) the Wechsler-Bellevue scale, the Henmon-Nelson test of mental ability, the Shipley-Hartford conceptual quotient scale, the Minnesota multiphasic personality inventory, and the Rorschach test.

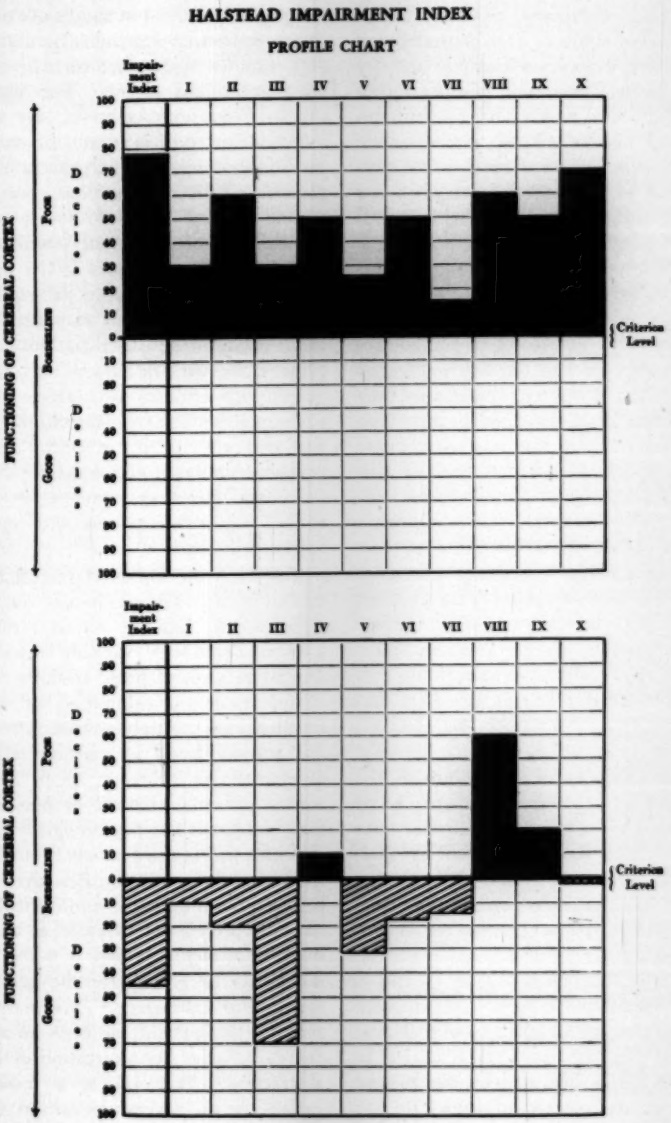

Fig. 3 and 4. Graphic presentation of mean performances of Halstead's frontal and non-frontal lobectomy groups.

The patient's performance on Halstead's battery indicates severe impairment of biological intelligence. These results are presented in graphic form in figure 2.

The profile chart (fig. 2) was constructed in Halstead's laboratory on the basis of a large group (N=451) composed of both brain-injured and control subjects, each person being individually tested over approximately a 2-day period. The heavy, horizontal midline represents the criterion level which best differentiates the control and brain-damaged performances. A score above this line represents a test performance which is similar to those given by brain-damaged subjects, and a score below is in the control range. Deciles above and below the criterion level are indicated along the ordinate (vertical axis). A score of 90 in the control range would indicate that the subject has done better than 90 percent of the control subjects. A score of 60 in the brain-damaged range would indicate a poorer performance than 60 percent of the brain-damaged group. Ten tests are indicated along the abscissa. These are the tests which Halstead found to differentiate significantly brain-damaged from control subjects. Since no single test is a perfect instrument in itself, Halstead feels on the basis of his results that a subject's performance should be in the brain-damaged range (above the criterion level) on at least 5 tests before intellectual impairment due to brain damage is assumed. The Halstead Impairment index is indicated on the extreme left of the chart, and represents a summary value for the ten discriminating tests.

Prior to consideration of the nature of the impairment indicated in Figure 2, it may be of interest to compare the chart with similar ones based on the mean scores obtained by Halstead's frontal and non-frontal lobectomies, charting the mean values which he published in his book (18). These graphs are shown in figures 3 and 4.

It will be observed from figures 3 and 4 that Halstead found much greater impairment of intellectual functions in patients with frontal lobectomies than in non-frontal lobectomies. In comparing the results obtained by our patient with agenesis of the corpus callosum (fig. 2) with those presented in figures 3 and 4, it is apparent that this patient's test performance was generally very similar to the scores obtained by the frontal lobectomy patients, exceptions occurring on two of the ten tests.

A more detailed analysis of these results requires identification of the ten tests involved. Test I on the chart represents the Halstead category test. This test makes use of a multiple-choice projection apparatus by means of which 208 stimulus figures are successively presented on a milk-glass screen. The 208 items are divided into seven subtests. By varying size, shape, color, position, etc., of the test items, the item content is changed as the test progresses. Each subtest has been organized around a single principle (such as uniqueness of position, color, size, etc.) which makes possible "correct" responses. If the subject is able to abstract the underlying principle and use it upon which to base his responses in a subtest even though the item content changes, his error score will be low. This test then requires such abilities as analysis and synthesis, differentiation and generalization. Our patient was deficient in this respect. This result is probably similar to the deficiency in abstract or conceptual thinking reported by Goldensohn, *et al.* (16) in their patient with agenesis of the corpus callosum.

Test II is the low-level critical flicker-fusion measurement. The apparatus uses a modified Type 631-B General Radio Strobotac as an electronic source of variably intermittent light, housed in a sound-proof container. It has provision for rapid calibration of intensity and frequency of the light source. On this measure our subject fused at an unusually high frequency of intermittency, much over the mean values for either frontal or non-frontal lobectomy patients. The psychological significance of this measure is open to question, but Halstead has found the result to differentiate significantly between brain-damaged and control groups. He has interpreted the measure as an estimate of his power factor, which is taken to represent the power or energy for the basic intellectual function of abstraction. This interpretation, applied to our patient, suggests an abundance of energy or drive for expression of her impaired abstraction. Test III is a measure of the variability on successive critical flicker-fusion trials. On this test the patient showed an extremely limited range of variability, fusing

at very nearly the same frequency on successive trials. Her average deviation was only 0.4 of a cycle per second. Our patient showed the same machine-like precision in this test as is common in frontal lobectomy. Tests IV, V, and VI represent the scores obtained from use of the Halstead tactual performance test. In this test a modification of the Seguin-Goddard form board is used. The subject is blindfolded, and is not permitted to see the form board or blocks at any time. The subject fits the blocks into their proper places with his dominant hand, then repeats the procedure with his other hand, and finally performs the task a third time using both hands. After the board and blocks have been removed, the blindfold is removed and the subject is required to draw a diagram of the board representing the blocks in their proper places. The subject is scored for the total time needed to place the blocks, for the number of blocks correctly reproduced, and for the number correctly localized in his drawing. Although components of this test contribute to Halstead's abstraction factor of biological intelligence, it is of particular interest with our patient to observe possible transfer effects from the performance with the right hand to the left. The subject required 44.7 minutes to place the blocks. The criterion point separating "normal" and "brain damaged" performances is 15.7 minutes, placing the patient well into the brain-damaged range. In nearly all instances the first performance (dominant hand) takes the longest time, the second performance (minor hand) the next, and the task is performed most quickly on the third performance when both hands are used. Our patient did not conform to this pattern. She needed 16.3 minutes with her right hand, and then upon repeating exactly the same procedure with her left hand, required 20.1 minutes to place the blocks. The third trial (both hands) required 8.3 minutes. These results suggest that no bilateral transfer occurred; that the patient profited not at all when using her left hand from the experience with the right. Since our review of the literature showed no other studies of bilateral transfer in agenesis of the corpus callosum, this result may be taken as suggestive that bilateral transfer may be impaired. Nevertheless, coordinated use of the hands was obvious to the observer as well as being implied by the shorter time needed on the third trial when both hands were used. In spite of requiring a total of 44.7 minutes to place the blocks, our subject was able to reproduce and localize fewer forms in her drawing than control subjects who are exposed to the board, on the average, less than one-third as long. Tests VII and VIII represent the rhythm subtest of the Seashore measures of musical talent and the speech-sounds perception test, respectively. Our patient's performance on these tests of auditory perception was closely comparable with the average results obtained by Halstead from his frontal lobectomy group. Test IX is the Halstead finger-oscillation test, which measures speed of tapping with the index finger of the dominant hand. Our patient was able to tap an average of only 27 times in a ten-second period, considerably under the mean of 43.9 found by Halstead for frontal-lobectomy patients. The final measure, Test X, is the Halstead time-sense memory test. This test involves the estimation of ten-second intervals following a series of trials in which the subject has observed the hand of an electric clock revolve ten times in ten seconds. The error shown by our patient in estimating these intervals was in the "normal" range, considerably less than either the frontal or non-frontal lobectomy groups of Halstead's. Although the memory portion of this test was well performed, the patient showed impaired visuo-motor coordination of gross movements as indicated by her poor performance in stopping the manually controlled movement of the clock's sweep second-hand while she was doing the preliminary trials with the use of vision. Her failure in this test was also attributable in part to difficulty in sustaining concentrated attention to the task for the series of ten-second intervals.

Two measures of psychometric intelligence, the Wechsler-Bellevue scale and the Henmon-Nelson test, yielded intelligence quotients of 61 and 60, respectively. The inter-test scatter on the Wechsler-Bellevue scale was not characteristic of brain damage. This is of interest in relation to the strong positive indication given by Halstead's measures of biological intelligence. The verbal intelligence quotient was 66 on the Wechsler-Bellevue and the performance quotient, 65. The Shipley-Hartford conceptual quotient scale yielded a conceptual

quotient of 83, which Shipley (42) classifies in the "moderately suspicious" range. This test compares vocabulary with abstraction items, and a low quotient is obtained when the vocabulary score is the higher of the two. It may be noted, again, that the Halstead category test, which measures abstract and conceptual thinking ability, described definite impairment in our patient. The Halstead-Wepman aphasia screening test showed no symptoms of dysphasia or related losses in our patient.

Personality measures included the Minnesota multiphasic personality inventory, the Rorschach test and the Halstead schematic face test. The MMPI profile was characterized by high scores on all scales except the masculinity-feminity scale, which showed normal female interests. The scores for hypochondriasis, depression, hysteria, psychasthenia, and schizophrenia were all above 70, more than two standard deviations above the mean value for the standardization group. This profile would suggest that the patient was beset by depression and anxiety, in addition to being rather generally emotionally unstable. The Rorschach was characterized primarily by indications of organic impairment, including many of the signs that have been described elsewhere (1, 5). The patient gave only 11 responses to the ten ink-blots, and most of these were vague impressions which she was unable to define clearly. Judging from the Rorschach scores which relate particularly to Halstead's abstraction factor of biological intelligence, it would seem that impaired abstructive ability is the primary feature of the Rorschach protocol. The record was too meagre to justify any pointed attempt toward description of dynamic personality factors. The Halstead Schematic Faces Test consists of nine schematic faces selected from the series developed by Brunswik and Reiter (43), which are presented in different groupings on two separate cards. The subjects is presented with a list of characteristics representing polar extremes, such as "gay," and "sad," "beautiful," and "ugly," and asked to select the face which best represents each characteristic. Halstead (44) has found from studying large groups of brain-damaged, mentally ill, and control subjects that each group shows remarkable consistency (far above chance expectancy) in selecting certain schematic faces for pleasant characteristics and certain other faces for unpleasant characteristics. Halstead (44) cited this result as an illustration of categorizing toward an irrational solution in contrast to the grouping behavior of his category test which requires an insightful grasp of principles. Our patient followed the general pattern in selecting the faces usually associated with unpleasant characteristics, but had no more than a chance number of "pleasant" faces for the desirable characteristics. This extremely unusual result suggests that our patient had not developed normally even in certain respects of categorizing not based on rational principles, perhaps as a result of her congenital brain anomaly.

A question may be posed concerning the extent to which internal hydrocephalus and maldevelopment of the cerebellum in this patient were responsible for the impaired test performances. This question is impossible to answer satisfactorily, since we have inadequate evidence of the possible effects of these factors on the test results. We have had an opportunity to administer the same battery of tests, however, to one patient with a cerebellar tumor and another with a tumor of the aqueduct of Sylvius. In each of these patients the ventricles were markedly dilated. The patient with the cerebellar tumor had an obvious ataxia of the upper extremities, and the other patient was nauseated and vomited at intervals throughout the testing. Each of these patients gave performances which were well within the normal range as compared to patients with cerebral cortical damage. Since our patient's intracranial hypertension had subsided following surgery, and she was not tested until three months after surgery, it would seem likely that this factor was not responsible. Ataxia would also seem unlikely since our patient with a cerebellar tumor scored in the normal range. While we have this evidence that ataxia and internal hydrocephalus do not necessarily cause a poor performance, we have no assurance that they are to be ruled out completely. It would seem, however, that agenesis of the corpus collosum was the primary factor in influencing the test results.

Summary and Conclusions

A review of the literature concerned with the relation of the corpus callosum to mental

functions reveals a good deal of diversity in conclusions depending upon the clinical material studied. Investigators of the effects of callosal tumors generally describe associated mental changes which are severe and extensive. Surgical section of the corpus callosum has yielded results which are largely negative. Study of patients with agenesis of the corpus collosum has indicated that intelligence may vary from normality to low levels, depending upon the extent of involvement of associated structures.

This paper presents the first study of a patient with congenital agenesis of the corpus callosum using standardized and quantified psychological tests validated for measuring the effects of brain damage. While generalization from a single case is unjustified, the results may be taken to indicate changes which may possibly be associated with this condition. A summary of the psychological findings for this 19 year old girl includes 1) severe impairment of biological intelligence, particularly of the Abstraction factor but with sparing of the Power factor, 2) an intelligence quotient in the moronic range, 3) deficiency in bilateral transfer, 4) impaired visuo-motor coordination, 5) impaired ability to sustain concentrated attention to a task for 10-second intervals, and 6) depression, anxiety, and emotional instability.

BIBLIOGRAPHY

(1) Aita, J. A., Reitan, R. M., and Ruth, J. M.: Rorschach's test as a diagnostic aid in brain injury. *Am. J. Psychiat., 103:* 770, 1947.

(2) Reitan, R. M.: Intellectual functions of aphasic and non-aphasic brain-injured subjects. *Neurology, 3:* 202, 1953.

(3) Reitan, R. M.: Intellectual functions in myxedema. *Arch. Neurol. & Psychiat., 69:* 436, 1953.

(4) Reitan, R. M.: Intellectual and affective functions in chronic brucellosis. *Am. J. Psychiat., 110:* 19, 1953.

(5) Piotrowski, Z.: Rorschach ink-blot method in organic disturbances of central nervous system. *J. Nerv. & Ment. Dis., 86:* 525, 1937.

(6) Harrower-Erickson, M. R.: Personality changes accompanying cerebral lesions: Rorschach studies of patients with cerebral tumors. *Arch. Neurol. & Psychiat., 43:* 859, 1940.

(7) Aita, J. A., Armitage, S. G., Reitan, R. M., and Rabinovitz, A.: The use of certain psychological tests in the evaluation of brain injury. *J. Gen. Psychol., 37:* 25, 1947.

(8) Reitan, R. M.: Intelligence and language functions in dysphasic patients. *Dis. Nerv. System, 15:* 131, 1954.

(9) Baker, R. C., and Graves, G. L.: Partial agenesis of the corpus callosum. *Arch. Neurol. & Psychiat., 29:* 1054, 1933.

(10) Bruce, A.: On the absence of the corpus callosum in the human brain, with the description of a new case. *Brain, 12:* 171, 1889.

(11) Cameron, J. L., and Nicholls, A.: Two rare abnormalities occurring in the same subject: partial absence of the corpus callosum; the stomach situated entirely within the thorax. *Canad. M. A. J., 11:* 448, 1921.

(12) Cass, A. B., and Reeves, D. L.: Partial agenesis; diagnosed by ventriculographic examination. *Arch. Surg., 39:* 667, 1939.

(13) Dandy, W. E.: Operative experiences in cases of pineal tumors. *Arch. Surg., 33:* 19, 1936.

(14) Dandy, W. E.: Congenital cerebral cysts of the cavum septi pellucidi (fifth ventricle) and cavum vergae (sixth ventricle): diagnosis and treatment. *Arch. Neurol. & Psychiat., 25:* 44, 1931.

(15) Davidoff, L. M., and Dyke, G. A.: Agenesis of the Corpus Callosum: diagnosis by encephalography; report on three cases. *Am. J. Roentgenol., 32:* 1, 1934.

(16) Goldensohn, L. N., Clardy, E. R., and Levine, K.: Agenesis of the corpus callosum: report of a case with neuropsychiatric, psychologic, electroencephalographic, and pneumonencephalographic studies. *J. Nerv. & Ment. Dis 93:* 567, 1941.

(17) Gowan, L. R., and Masten, M. A.: Agenesis of the corpus callosum: diagnosis of a case by encephalography. *Am. J. Dis. Child., 60:* 1381, 1940.

(18) Halstead, W. C.: *Brain and Intelligence.* Chicago: Univ. of Chicago Press, 1947.

(19) Hebb. D. O.: Intelligence in man after removal of cerebral tissue: report of 4 left frontal lobe cases. *J. Gen. Psychol., 21:* 73, 1939.

(20) Brickner, R. M.: Brain of patient A. after bilateral frontal lobectomy; status of frontal lobe problem. *Arch. Neurol. & Psychiat., 68:* 293, 1952.

(21) Hyndman, O. R., and Penfield, W.: Agenesis of the corpus callosum: its recognition by ventriculography. *Arch. Neurol. & Psychiat., 37:* 1251, 1937.

(22) Ironside, R., and Guttmacker, M.: Corpus callosum and its tumors. *Brain, 52:* 442, 1929.

(23) Voris, H. C., and Adson, A. W.: Tumors of the corpus callosum; a pathologic and clinical study. *Arch. Neurol. & Psychiat., 34:* 965, 1935.

(24) Alpers, B. J., and Grant, F. C.: Clinical syndrome of the corpus callosum. *Arch. Neurol. & Psychiat., 25:* 67, 1931.

(25) Alpers, B. J.: A note on the mental syndrome of corpus callosum tumors. *J. Nerv. & Ment. Dis., 84:* 621, 1936.

(26) Sweet, W. H.: Seeping intracranial aneurysm simulating neoplasm: syndrome of corpus callosum. *Arch. Neurol. & Psychiat., 45:* 86, 1941.

(27) Collier, J.: Localization of function in the nervous system. *Brit. Med. J., 1:* 55, 1930.

(28) Kirschbaum, W. R.: Agenesis of the corpus callosum and associated malformations. *J. Neuropath. & Exper. Neurol., 6:* 78, 1947.

(29) Van Wagenen, W. P., and Herren, R. Y.: Surgical division of commissural pathways in the corpus callosum: relation to spread of an epileptic attack. *Arch. Neurol. & Psychiat., 44:* 740, 1940.

(30) Smith, K. U., and Akelaitis, A. J.: Studies on the corpus callosum. I. Laterality in behavior and bilateral motor organization in man before and after section of the corpus callosum. *Arch. Neurol. & Psychiat., 47:* 519, 1942.

(31) Akelaitis, A. J: Studies on the corpus callosum. II. The higher visual functions in each homonymous field following complete section of the corpus callosum. *Arch. Neurol. & Psychiat.,* 45: 788, 1941.

(32) Akelaitis, A. J., Risteen, W. A., Herren, R. Y., and Van Wagenen, W. P.: Studies on the corpus callosum. III. Dyspraxia and apraxia following partial and complete section. *Arch. Neurol. & Psychiat.,* 47: 971, 1942.

(33) Akelaitis, A. J.: Studies on the corpus callosum. IV. Diagnostic dyspraxia in epileptics following partial and complete section of the corpus callosum. *Am. J. Psychiat., 101:* 594, 1945.

(34) Akelaitis, A. J.: Studies on the corpus callosum. V. Homonymous defects for color, object and letter recognition (homonymous hemiamblyopia) before and after section of the corpus callosum. *Arch. Neurol. & Psychiat., 48:* 108, 1942.

(35) Akelaitis, A. J.: Studies on the corpus callosum. VI. Orientation (temporal-spatial agnosia) following section of the corpus callosum. *Arch. Neurol. & Psychiat., 48:* 914, 1942.

(36) Akelaitis, A. J.: Studies on the corpus callosum. VII. Study of language functions (tactile and visual lexia and graphia) unilaterally following section of the corpus callosum. *J. Neuropath. & Exper. Neurol., 2:* 226, 1943.

(37) Akelaitis, A. J.: Studies on the corpus callosum. VIII. Effects of partial and complete section of the corpus callosum on psychopathic epileptics. *Am. J. Psychiat., 98:* 409, 1941.

(38) Akelaitis, A. J.: Study of gnosis, praxis and language following section of the corpus callosum and anterior commissure. *J. Neurosurg., 1:* 94, 1944.

(39) Bridgman, C. S., and Smith, K. U.: Bilateral neural integration in visual perception after section of the corpus callosum. *J. Comp. Neurol., 83:* 57, 1945.

(40) Parsons, F. H.: Eight cases of section of corpus callosum in individuals with a history of epileptic seizures: psychological tests. *J. Gen. Psychol., 29:* 227, 1943.

(41) Smith, K. U.: Experimental analysis of the associative mechanism of the human brain in learning functions. *J. Comp. & Physiol., Psychol., 45:* 66, 1952.

(42) Shipley, W. C.: A self-administering scale for measuring intellectual impairment and deterioration. *J. Psychol., 9:* 371, 1940.

(43) Brunswik, E., and Reiter, L.: Eindruckscharactere schematisierter Gesichter. *Ztschr. Psychol., 142:* 67, 1937.

(44) Halstead, W. C.: Biological intelligence. *J. Personality, 20:* 118, 1951.

LATENT EPILEPSY AS A FACTOR IN TENSION STATES

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Pathological tension is present as a symptom in many psychiatric conditions. Normally it occurs as an organismal response to a life situation. People vary, according to their constitutions and life experiences, in the way in which they respond to stress. Dissociation, obsessional rituals, impulsive outbursts and epileptic fits are but some of the ways in which people react, while some respond merely by diffuse anxiety. The latter form an etiologically mixed group in which tension is the leading symptom. These patients are frequently diagnosed as "tension states." These states are marked by multiple symptomatology. The patients complain of "feeling on edge," "being unable to relax," restlessness, anxiety, of their "thoughts going round," insomnia, headaches, irritability and exhaustion. There is a tendency to somatize symptoms, but apart from this the patient may obviously be losing weight. Numerous methods of treatment have recently been described. Mephenesin, hexomethonium, chlorpromazine, somnifaine narcosis, acetylcholine shock, modified insulin therapy and cortical undercutting may be quoted as examples. Some cases, however, prove very resistant to treatment and any information of an etiological character, which may ultimately throw light on a rational therapy, is to be welcomed. This paper proposes to show that some "tension states" are in fact cases of epilepsy, possibly latent at the time of examination. In this respect it should be noted that the epileptic prodrome is a "tension state" with symptoms similar to those described above. The prodrome usually lasts a day or so and is relieved by the epileptic discharge, while in the cases under review this discharge failed to occur and tension continued for weeks or months. Like the psychogenic "tension state," the prodrome may also be a response to stress. In epilepsy, however, the brain is liable to paroxysmal discharges. This disturbed neurophysiology may be clearly demonstrated by the EEG. The prodrome should thus be viewed as a result of combined external stresses and internal instability. In some cases environmental factors and in others the central instability play the dominant rôle.

We regret that it is impossible to say how frequently latent epilepsy is a factor in "tension states." One reason is that the condition is usually treated on an out-patient basis, without full investigation. Only the severer cases are admitted to a mental hospital and more fully investigated. Since the connection was first observed, however, more cases of tension have been investigated and the relationship appears to be met with not infrequently. The EEG is of paramount importance in establishing a complete diagnosis and it has been methodologically easier for us to obtain retrospective evidence of epilepsy from our EEG records than from our clinical records.

The finding which first drew our attention to the possibility that latent epilepsy formed a possible basis of tension states, was the 3rd or lower subharmonic response to photic stimulation, which, in our view, may be an indication of latent epilepsy, even in the presence of an otherwise normal record. In order to demonstrate this, all responses to photic stimulation were reviewed. It was then possible to show that 3rd or lower subharmonics were usually associated with further evidence of epilepsy in the EEG. When subharmonics occurred alone they were further investigated. These patients, as will be seen, showed tension as a prominent symptom. An automatic analyzer was used which made the recognition of subharmonics an easy task.

EVIDENCE OF THE SIGNIFICANCE OF 3RD SUBHARMONIC RESPONSES

A subharmonic is, following Mundy-Castle (4), an evoked rhythm which is a fraction of the fundamental stimulus frequency. These were classified as 2nd or 3rd subharmonics if the response was ½ or 1/3 of the fundamental flash rate respectively: e.g., if the flash

rate was 18f/s with the stroboscope, a response of 9c/s in the EEG record is a 2nd subharmonic, while a 6c/s response in the EEG is a 3rd subharmonic. In this paper subharmonics lower than the 3rd subharmonic (e.g. 4th, 5th, etc.) have been classified with 3rd subharmonic responses. Walter (9) states that the most striking feature of photic stimulation of epileptics is the generation of subharmonics which he feels are probably pathological. In fact Walter and Walter (8) state that subharmonic responses to photic stimulation often precede the appearance of a characteristic seizure pattern.

Mundy-Castle (4) studied two series of normal adults, divided according to age into young and old groups. He found that the incidence of 2nd subharmonics was significantly lower among the older age group as compared with the younger group, while the older group showed a significantly greater incidence of 3rd subharmonics. No sex difference was noted. He found that 3rd subharmonics were rare and usually associated with abnormal features, such as typical polyspike discharge or wave and spike response. Thus, the 3rd subharmonic response seemed to be associated with epileptic manifestations and Walter and Walter (8) found a high proportion of subharmonic activity among evoked seizures of the wave and spike type.

Mundy-Castle (5) comparing a probable epileptic group to normals found a significantly higher incidence of 3rd subharmonic responses to photic stimulation in the abnormal group. This, however, was not the case among a group of Africans who suffered chiefly from major epilepsy or other types of seizures comparable to psychomotor epilepsy.

TABLE 1.—Showing Relationship of Subharmonics to Sex of Patients and Distribution of Subharmonic Responses.

	2nd subharmonics	*3rd subharmonics*	*3rd subharmonics and spikes*
Male	11	11	2
Female	13	12	17
Total	24	23	19

For n = 2, $\chi^2 = 7.72$ (significant at 5 per cent level of confidence).

Further Evidence on the Significance of 3rd Subharmonic Responses

The Electroencephalographic Department at Runwell Hospital undertakes about 500 examinations each year, which means that since photic stimulation with the Scophony-Baird Stroboscope was introduced just over three years ago, some 1,600 records have been made. After excluding 10 patients under 15 years of age, it was found that 66 patients (approximately 4 per cent of all patients examined) showed 2nd subharmonics, 3rd or lower subharmonics, spikes, or a combination of these in response to photic stimulation. Twenty-four patients had 2nd subharmonics, 23 had 3rd or lower subharmonics and 19 had 3rd or lower subharmonics and spikes. In the total of 19 patients showing spike responses are included 7 who showed a typical spike and wave response (see table 1).

The sex distribution of the patients was examined and showed no sex difference except that when spikes were elicited, women predominated (see table 1).

There is no significant age difference in the groups, although patients with 2nd subharmonics gave a slightly lower mean age than patients with 3rd subharmonics and spikes.

The 66 EEG resting records were classified as "within normal limits" or "abnormal" and, when related to the type of subharmonic or the occurrence of spikes, certain trends became evident. First, when 2nd subharmonics only were present, normal resting records were found more frequently than abnormal records. Where 3rd or lower subharmonics occurred in any combination, abnormal records out-numbered normal resting records. The same was even more clearly evident when spikes occurred with 3rd subharmonics (see table 2). Thus,

TABLE 2.—Showing Relationship of Subharmonics to the EEG Resting Record.

	Normal EEG	*Abnormal EEG*	*Total*
2nd subharmonics	18	6	24
3rd subharmonics	10	13	23
3rd subharmonics and spikes	4	15	19

For n = 2, $\chi^2 = 12.7$ (significant at 1 per cent level of confidence).

TABLE 3.—SHOWING EEG DIAGNOSIS IN THE ABNORMAL EEG RESTING RECORDS.

	Epilepsy	*Non-specific*	*Total*
2nd subharmonics	2	4	6
3rd subharmonics	12	1	13
3rd subharmonics and spikes	15	0	15

For $n = 2$, $\chi^2 = 13.2$ (significant at 1 per cent level of confidence).

3rd or lower subharmonics, especially when associated with spikes, frequently occurred with abnormal EEGs. This fact was further exemplified by examining the occurrence of subharmonics and spikes in relation to the final EEG diagnosis (see table 3). Epilepsy was revealed in this group as the commonest abnormal cause. This was especially so when the 3rd subharmonics fell in the theta band of 4–7c/s.

Normally subharmonics occurred symmetrically on both sides of the brain, but occasionally they were unilateral. In one case, a cerebral tumor was associated with epilepsy of the Jacksonian type. Here, both the abnormality in the resting record and the 3rd subharmonic were unilateral and on the same side as the lesion. In another case diagnosed as epilepsy, a lesion was queried in the left hemisphere, as the paroxysmal activity in the resting record was asymmetrical. Here, again, the 3rd subharmonic was predominantly on the abnormal side.

It has been routine practice to discontinue photic stimulation as soon as abnormality was apparent on the EEG record. In only two cases was a grand mal reported following photic stimulation, and in both of these 3rd and lower subharmonics preceded spikes, which were in turn followed by the convulsion. Finally, patients who responded to photic stimulation with spikes, when re-examined after taking anticonvulsants, showed only 3rd and lower subharmonic responses and in some cases even these were abolished.

Investigation of the Clinician's diagnosis (see table 4) revealed further interesting information. 2nd subharmonics were seen to be associated with a diagnosis of immature and inadequate personality, often qualified by the adjective hysterical, while 3rd and lower subharmonics, with or without spikes, were associated with a diagnosis of epilepsy per se or "tension state."

On the whole these findings bear out Mundy-Castle's observations (4, 5). The results which we wish to stress are the association of 3rd and lower subharmonics:

(a) with spikes,
(b) with abnormal resting records,
(c) with frequent EEG diagnosis of epilepsy,
(d) with frequent clinical diagnosis of epilepsy or "tension state,"
(e) in unilateral association with unilateral EEG abnormality,
(f) as the antecedents of spikes prior to anticonvulsants, and a possible persisting response after anticonvulsants.

In our view these findings give a significance to 3rd or lower subharmonic responses, even when they occur alone in a normal record. They also point to a further possible refinement in the EEG diagnosis of epilepsy. The 3rd or lower subharmonic may be seen in treated and controlled cases, or as a minimal

TABLE 4.—SHOWING CLINICAL DIAGNOSIS OF THE SUBHARMONIC GROUP ON REFERRAL FOR EEG.

	Epilepsy	*Organic*	*Tension*	*Immature or Inadequate Personality Reaction*	*Total*
2nd subharmonics	2	3	3	16	24
3rd subharmonics	7	2	10	4	23
3rd subharmonics and spikes	8	2	8	1	19

For $n = 3$, $\chi^2 = 23.36$ (significant at > 0.1 per cent level of confidence). 3rd subharmonics, and 3rd subharmonics and spikes are treated together.

sign of latent epilepsy. The desirability, then, of further investigating any 3rd subharmonic response is apparent.

Clinical Aspects.

Brain (1) mentions epileptics who have one or perhaps two fits during their whole lifetime and Walshe (7) says that "cases are known in which after one or more fits the affliction disappears finally." The epileptics to whom we now wish to draw attention were in the obscure group, where a frank history of fits was by no means clinically clear and where, in our view, the prodrome, by giving rise to a prolonged state of tension, was of great importance. A normal resting EEG is clearly no longer the end of the matter and the illustrative cases presented were drawn from the EEG group where a 3rd or lower subharmonic response to photic stimulation was the only irregular finding at routine examination. These cases may be classified into four types:

1. Where up to the time of EEG examination no fits had occurred, but where epilepsy later became apparent.
2. Where the patient appeared to be a "cured epileptic," i.e. there was an early history of fits, but for a considerable time, without control, the patients had been fit-free.
3. Where a history of a fit under special circumstances or under special stress was obtained.
4. Where no history of fits was obtained and fits have not yet occurred.

Case 1. "Tension state" with subsequent onset of epilepsy.

In July 1953 Mrs. R. Y., aged 36, presented complaining of depression, headaches and "a feeling that she could scream." She was described as "strung up and hysterical." The patient was an illegitimate child. She was adopted, when a few weeks old, by a childless couple who, when she was 3, had a child of their own. At about that time the family moved from the country to London, leaving patient with her adoptive grandmother to "keep her company," and there patient remained until 14 years, with annual visits from her adoptive mother and step-siblings. Patient entered domestic service at 14 years and then went to London to work in a shop and live with her adoptive mother. After a short while she moved into rooms as "she wanted to be independent." She became engaged at 19 and shortly afterwards was thrown from her bicycle when knocked down by a car. She was unconscious for 40 minutes. While she was in hospital her fiancé quarrelled with her adoptive parents, and in loyalty to him patient did not see them for about ten years. She married and had two children, a girl of 13 who suffered from epilepsy and a boy of 7. Her marriage has been unhappy and marred by quarrels, hysterical behavior on both her own and her husband's part, and she has now left home. She has had one long but frustrating love affair with a married man, who finally decided to stay with his wife. Her first appearance at the Out-patient Clinic was at a time when her quarrels at home were reaching their climax. There was then no history of fits or blackouts. The patient failed to respond to superficial psychotherapy and a holiday. An EEG was performed in August 1953, in view of her daughter's history. The resting record was slightly abnormal but the response to photic stimulation was dramatic; she complained of severe tension and screamed. Next, myoclonic movements appeared and finally she complained of visual hallucinations, the faces of acquaintances "came crowding in on her." These complaints were associated with the appearance of 3rd subharmonics, followed by spikes in the EEG (fig. 1). In December 1953, 3 months after her first EEG examination, the patient had a grand mal. In January 1954 she had a second grand mal and was then admitted to hospital. EEG examination was again performed and photic stimulation was now followed first by an exacerbation of tension and finally by a fit. She was treated by phenobarbitone, gr. i. t.d.s., and photic stimulation no longer disturbed her. The EEG record now showed only a 3rd subharmonic response (fig. 2). She has now left her husband and is living alone. She works full time as a shop assistant. Her tension has abated remarkably and she has no further fits.

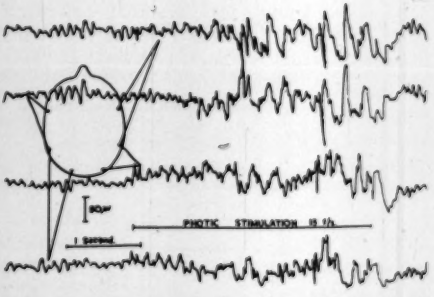

Fig. 1. Case. 1. Initial response to photic stimulation showing 3rd and lower subharmonics mixed with spikes, which are maximal anteriorly and symmetrical on the two sides.

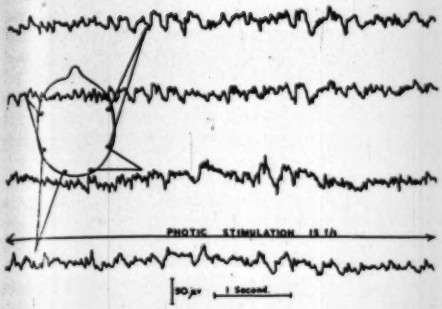

FIG. 2. Case 1. Showing that after a small dose of phenobarbitone the spikes were abolished, but the subharmonics remained unchanged.

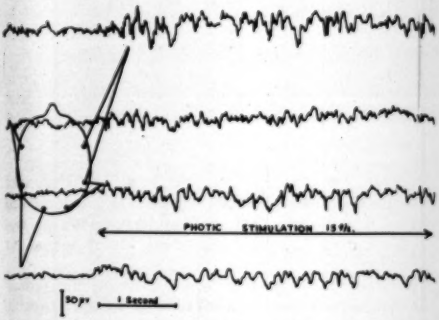

FIG. 3. Case 2. Response to photic stimulation showing 3rd and lower subharmonics with spikes, chiefly on the right side.

Case 2. Spontaneously cured epilepsy.

Mr. M., aged 25 years, was admitted to hospital following a serious attempt at suicide. He was the youngest of 6 siblings born of healthy parents. His father was a small tradesman. There was no family history of any nervous or mental disease. Both his parents died of carcinoma. His early years and development were normal. At school, he was an average scholar and mixed easily with his schoolmates. His work record is steady and without note.

At the age of 15 years he had a series of "faints" preceded by giddiness. Treatment at that time was limited to reassurance as the faints were thought to be secondary to the fact that his mother was in hospital dying of cancer. This event had brought about the first separation from his mother.

His life continued uneventfully until 3 years ago, when, at the age of 22 years, he married. A year later his wife gave birth to a stillborn child. Patient was inordinately upset by this. He commenced to brood and headaches developed. He became increasingly irritable and without reason quarrelled with his wife. Normally, he was a placid, contented man. He became excessively worried over his work and felt it was too much for him. At night he was restless, troubled by recurring thoughts and unable to compose himself for sleep. His appetite became impaired and he lost weight. This increasing tension culminated in a violent quarrel with his wife and was followed by a suicidal attempt, when he took a number of sleeping tablets. He was unconscious for two days. During the period of recovery he had two major convulsions. A diagnosis of reactive depression in a chronic tension state was made after recovery. This diagnosis was confirmed by projection tests. However, in view of his faints and the two fits on recovering from barbiturate poisoning, latent epilepsy as a cause of his tension was considered.

An EEG examination revealed a normal resting record. However, photic stimulation produced 3rd and lower subharmonic responses and occasional sharp waves and spikes. The spikes appeared chiefly on the right side. Stimulation at 15f/s elicited an irregular response at 5c/s with some spikes (fig. 3). When the stimulation ceased the trace continued at 5c/s and then the amplitude of the waves gradually increased while the frequency dropped so that 20 seconds later the rate was 2-4c/s (fig. 4). 70 seconds after the end of photic stimulation the record had become regular at about 3c/s. The amplitude of the waves at this stage had increased from 50 to 150uV (fig. 5). High voltage sharp waves were then appearing, chiefly on the left side. This regular rhythmic discharge continued for a further 2½ minutes until a major convulsion occurred.

While this record was obtained the patient became increasingly tense and restless. After the convulsion he was better, but within a few days his tension had recurred. He was put on mysoline therapy which resulted in a dramatic relief of all his symptoms. Previously it was discovered that subharmonic spike responses were completely suppressed by phenobarbitone. Mysoline, in preference to phenobarbitone, was used in treatment as it is claimed that its sedative action is minimal. In this case the response to therapy may be considered as

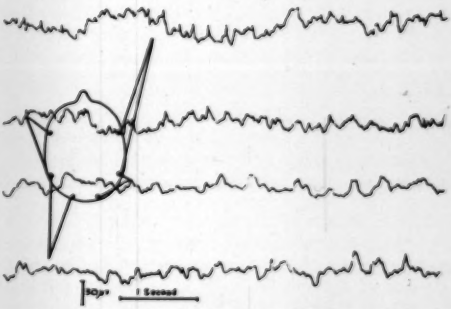

FIG. 4. Case 2. Showing persistent low frequency discharge 20 seconds after cessation of photic stimulation.

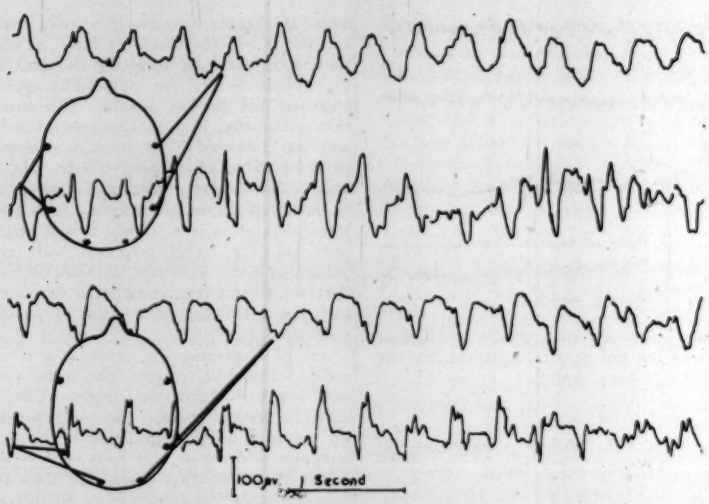

Fig. 5. Case 2. 70 seconds after the end of photic stimulation showing regular slow activity at 3c/s with high voltage sharp waves which are chiefly on the left side. The amplitude has increased from 50 to 150 uV.

directly related to the control of his latent epilepsy, rather than to any non-specific damping down of tension by sedation.

Case 3. Fit under special stress.

Mr. U., aged 34, had a long history of alcoholism. He was an illegitimate child who was brought up in an orphanage. He joined the Army as a boy and first saw service in Dunkirk where he was badly bombed. He was evacuated to England and saw no more active service for four years, when he was sent to Italy. He was then under heavy shell fire. It is not clear whether he was concussed, but he was found in a dazed state and evacuated. Despite a psychiatric recommendation to the contrary, he was returned to the line, and within seven days had a hematemesis which necessitated his evacuation to England. After a period of medical treatment a partial gastrectomy was performed. His consumption of alcohol had increased insidiously from 1943 to 1944, and in 1949 he sought psychiatric advice. After several abortive attempts to deal with him as an out-patient, he was sent to a mental hospital in 1950. During this admission he was reported to have had one convulsion and this was attributed to the withdrawal of harbiturates which had just been carried out. After several more admissions to mental hospitals, he was finally treated at Runwell Hospital in 1953. A diagnosis of "psychopathic personality with alcoholism" was made. He said that only alcohol or barbiturate made life tolerable for him because of constant subjective tension.

An EEG showed a normal resting record and over-breathing response. Photic stimulation elicited 2nd and 3rd subharmonics. Injection of 3 cc. 5 per cent metrazol, which is 150 mg. metrazol, in the manner described below produced spikes in response to photic stimulation. The patient complained of unbearable tension which resembled his symptoms at their worst.

Treatment was started with ECT with the rationale that this tension resembled that of epileptics in the interparoxysmal phase. The patient often remarked that he was irritated by other patients and that it was a job to restrain himself from violence at times. For three months, with spaced ECT, the patient was free of tension or, at any rate, when this appeared it was relieved by a single treatment. He finally discharged himself after refusing further treatment and relapsed.

Case 4. No history of fits.

Mr. G. was a well-built man, aged 42, over 6 feet in height and weighing over fifteen stones. He came to the out-patient clinic complaining of tension, insomnia and depression. His story was that for over 20 years he had worked in the head office of a large bank. He was not a particularly good scholar and despite a Grammar School education had not matriculated. He had also not been able to pass the Banker's examination and consequently had been overlooked for promotion, so that people much younger than himself, and with less service at the bank, were working over him. He had originally been in the public part of the bank but had lost his temper with a customer and was moved to the counting house. He had frequently lost his temper in this department, and on one or two occasions had assaulted other clerks. He complained bitterly of the noise and jostling in the office. As far as his

personal life was concerned, he was married and had a child. His relationship with his wife was bad; they slept in separate rooms and were barely on speaking terms. During his RAF service he had suffered from "nerves," but these improved on transfer to a small isolated unit. He said he was really happy only when on the golf course.

His resting EEG was quite normal and there was no overbreathing response. Photic stimulation elicited a 3rd subharmonic response. He was injected intravenously with 2 cc. 5 per cent metrazol and the injection was continued at 1 cc/½ min. to a total of 4 cc., which is 200 mg. metrazol. Spikes now appeared in response to photic stimulation. At a later interview the patient remarked quite spontaneously that during the injection he felt just as he did when he lost his temper, and he was becoming so annoyed that he was about to assault the injector. He also asked whether he had said anything aloud as he was clouded for a short period of time. He complained of unbearable tension as a result of the injection.

A transfer was obtained for him to a quiet, rural branch where he has worked for six months without complaint.

Discussion

The first case presented as a tension state with a long history of stress. In this case the constitutional aspect was obvious as her daughter was epileptic. Her own complaints preceded her first fit by about 3 years. Photic stimulation alone reproduced her symptoms at their worst and when continued produced myoclonic movements, visual hallucinations or a fit. Phenobarbitone provided an adequate safeguard against this with some clinical improvement. A satisfactory social solution was found.

The second case exemplifies clearly the interaction of exogenous factors with a latent epileptic constitution in the production of tension. Once sufficient stress had occurred, tension seemed to be self-perpetuating. The stillbirth produced the initial anxiety and tension, which then continued in an increasing degree for 2 years. Photic stimulation also acted as a source of external stress. This produced a low-frequency dysrhythmia in the EEG. However, when the stress was removed the dysrhythmia continued and increased in amplitude in a self-perpetuating manner and was accompanied clinically with increasing uneasiness. A major fit eventually resulted, with temporary relief of tension.

The third case also complained of tension, which became comprehensible in similar terms. Here, however, the tension was continuous, and while the external environment must have played a large part in exciting it (early and war-time experiences), environmental manipulation offered little hope, for changes of jobs and admission to hospital had already no effect. A temporary improvement was produced by manipulation of the internal environment and indeed the patient had learned to do this himself with alcohol.

The last case was a tension state where, as a result of following the clue offered by the 3rd subharmonics, the psychological tension also became comprehensible in physical terms as an epileptic prodrome. The symptoms were produced by over-reaction to environment and abated with the move to a quiet, rural bank.

In the last two cases the metrazol flicker threshold was very low and 150 mg. in Case 3 and 200 mg. in Case 4 produced spikes. Leiberman, Hoenig and Haeker (3) found that there was a variable threshold to excitation of the EEG in a series of 124 neuropsychiatric cases. The threshold was lowest in idiopathic epilepsy and catatonic schizophrenia. The level in our two cases was about 1/3 of the mean threshold dosage of metrazol required to activate the EEG in these conditions. Four more recent cases of 3rd subharmonics have been investigated by metrazol activation alone. In these cases the threshold of activation of the EEG varied from 150 to 200 mg. This dosage was sufficient to produce spike and wave discharge. The threshold was below the critical level of 350 mg. found by Fuglsang-Frederiksen (2). Fuglsang-Frederiksen noted that only 1 out of 63 controls, but 79 out of 96 idiopathic epileptics, fell below this level.

The above facts, while not conclusive in themselves, strengthen the argument already put forward that 3rd subharmonics are indicative of epilepsy.

Ulett, Gleser Goldine, Winokur and Lawler (6), when investigating the EEG response to photic stimulation as an index to anxiety proneness, found that some individuals responded with symptoms of marked tension and organized illusions. "A few cases produced overt behavioral manifestations varying in degree from anger to convulsive seizures. In these cases slowing of the EEG record occurred in

response to photic stimulation. The authors posed the question as to whether these cases were in fact epilepsy. The similarity of their cases to those described in this paper is very close.

It is not possible to postulate any simple cause-effect relationship to explain these findings. Epilepsy is essentially a defect in the internal environment which, interacting with the external environment, results in prodromes or convulsions. The most frequent description of a prodrome based on the patient's subjective appreciations is that of "tension." "Tension" is merely the patient's description of his feelings, which once again result from a complex interaction. And the tension itself is not only apparent directly but also causes indirect effects in the course of the attempts by the patient's personality to contain it.

The physical treatment of epilepsy or real environmental manipulations may be useful in the treatment of this group of tension states. As a corrollary it should be noted that epilepsy may also be influenced by environmental changes. Many patients have fits only at a time of physical or emotional stress, menopause, puberty, menstruation and conversely admission to hospital with its protective atmosphere not infrequently abolishes fits for long periods.

Summary

1. The 3rd subharmonic and spike responses to photic stimulation are most frequently seen with an abnormal resting record and an EEG diagnosis of epilepsy.

2. Cases are described with 3rd subharmonic and lower responses to photic stimulation on the EEG, and with tension as a leading symptom.

3. The results of metrazol activation and metrazol flicker are described in a few cases and the low threshold of activation of the EEG noted.

4. The treatments of epilepsy or real material external changes may influence this group of "tension states."

Acknowledgments

We wish to express our thanks to Dr. R. Ström-Olsen, Physician Superintendent, Runwell Hospital, for permission to publish this paper and to Dr. W. P. Berrington for access to cases under his care. We should also like to thank Dr. G. A. Foulds for the statistical evaluations.

Bibliography

(1) Brain, W. R.: *Diseases of the Nervous System.* 3rd ed. London: Oxford University Press.

(2) Fuglsang-Frederiksen, V.: Activation of EEG disturbances with metrazol (pentazol) in epileptics, normals and patients with syncopal attacks. *Electroencephalog. & Clin. Neurophysiol., 4:* 471-480, 1952.

(3) Leiberman, D. M., Hoenig, J., and Haeker, M.: The metrazol flicker threshold in neuropsychiatric patients. *Electroencephalog. & Clin. Neurophysiol., 6:* 9-18, 1954.

(4) Mundy-Castle, A. C.: Analysis of central responses to photic stimulation in normal adults. *Electroencephalog. & Clin. Neurophysiol. 5:* 1, 1953.

(5) Mundy-Castle, A. C.: Clinical significance of photic stimulation. *Electroencephalog. & Clin. Neurophysiol., 5:* 187, 1953.

(6) Ulett, G. A., Gleser, Goldine, Winokur, G., and Lawler, A.: The EEG and reaction to photic stimulation as an index of anxiety-proneness. *Electroencephalog. & Clin. Neurophysiol., 5:* 23, 1953.

(7) Walshe, F. M. R. *Diseases of the Nervous System,* 7th ed. Baltimore: The Williams & Wilkins Co., 1952.

(8) Walter, V. G., and Walter, W. Grey: Effect of physical stimuli on EEG. *Electroencephalog. Clin. & Neurophysiol.,* Supp. No. 2, 1949. 2e Congrès Internationale d'Electroencéphalographie, Paris, p. 60.

(9) Walter, W. G.: Discussion on recent advances in the EEG diagnosis of epilepsy. *Proc. Roy. Soc. Med., 44:* 315, 1950.

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OVER-IDENTIFICATION IN THE SCHIZOPHRENIC CHILD AND ITS RELATIONSHIP TO TREATMENT[1]

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One of the schizophrenic child's most serious problems is the identification and delimitation of ego boundaries. Bender has written of the schizophrenic child's difficulties in relation to space and body image (2, 3). Gurevitz has defined ego identification as one of the aims of therapy with schizophrenic children. He has described methods of achieving this with the children he has treated (8, 9). Though manifesting the severe anxiety characteristic of schizophrenia, most of these children were autistic and detached. One of the first aims in his therapy is to establish in the patient the kind of excessive dependent over-identification with the therapist that so frequently exists in nature between schizophrenic and the significant adults upon whom they depend. In the over-identification, Gurevitz stipulates his next step is to delimit for the child the child's own boundaries. In the over-identification so often found in nature the significant adult either does not try or does not know how to induce ego identification in the schizophrenic child. Often an increasingly intense relationship of mutual dependency is established such as that described by H. S. Sullivan (10).

The problem of over-identification in schizophrenic children includes a consideration of 1) the nature of the process of over-identification and 2) the personality of the person with whom the child over-identifies. In therapy it is desirable to use these components constructively. The aim of this paper is to define the syndrome of over-identification in schizophrenic children, to describe some of the problems it presents in therapy and to suggest means of dealing with it constructively for the child.

Definition of Over-identification

By this term is meant here an intense emotional attachment to another person such that the other person is felt to be part of or co-existent with oneself or, perhaps more meaningfully in this paper, that one is but a part of the other person and would have no existence without the other. It may be considered as a form of parasitic or, at times, symbiotic relatedness in which the ego boundaries of both subjects are temporarily fused or lost to a greater or lesser degree. In infants, and possibly in their mothers too, for long periods of time it may be considered a healthy and constructive relationships (6). It has been described as occurring transiently in healthy adults in orgasm (1). Many fiction writers regard over-identification as pathognomonic of true love. Certainly it is a state of feeling found among others than child schizophrenics but among a certain type of the latter it is found more consistently and more pathologically than in other people. In itself it is not pathological and in the treatment of child schizophrenics it can be used constructively.

Manifestations of Over-identification

These will be discussed in terms of the degree of ego development and in terms of the type of relationship observed.

Ego development

The infant's perception of himself remains speculative. On the basis of infant behavior, it is postulated that in the first year the child gradually discovers body boundaries and the separateness of the mother(4). He is said to discover this by his inability completely to control his source of supply for body needs. By 18 months, when conscious sphincter control is first possible, the child is said to conceive of himself as an individual personality as well as a separate body in space(5). From then on, by trial and error in interaction with the environment and his own needs, the child develops patterns of behavior and incorporates attitudes of mind which though derived from the outside world are unique for himself when processed by his own developing ego.

[1] From the Children's Ward and the Children's Division of the Mental Hygiene Clinic of Bellevue Hospital, Psychiatric Division.

In the schizophrenic child this development of self awareness may be delayed or uneven or both, usually both. The schizophrenic child of three years or older may not know where his own body ends and the rest of the world begins. In children with this disability may be grouped those who demonstrate difficulty in identifying themselves in space. They spontaneously whirl and dance; they examine the surfaces of objects and people; they are preoccupied with growth and creativity; they test out the principles of gravity.

D. B., a 3½-year-old white boy, was brought to the hospital by his parents because of his rocking, headbanging, insomnia, and constant overactivity. He said three or four words to himself but otherwise did not speak. He was denuditive. He grimaced, whirled and was preoccupied with his own body, parts of which he examined with amazement as if they were not part of himself. After several weeks of psychotherapy, D. B. paid enough attention to the environment to try to telephone his mother. At this time too he began to show interest in the body of his therapist which he began examining and comparing part for part to his own body.

P. K. was an 11-year-old white boy who was preoccupied with processes of growth and change. He said he disliked drawing because the pictures were not real and thus would stay the same. He wanted to know how he could make live trees and people. He was preoccupied with birth, death and creativity.

Even if the schizophrenic child succeeds in determining his body boundaries, he has the greatest difficulty in clarifying his ego boundaries. He is beset by anxiety over self-assertion. In children with this disability may be grouped those who demonstrate difficulty in identifying themselves in relation to themselves as well as to others. Some of them are afraid of their own body functions and regard their body parts or excreta as having a separate existence often dangerous to themselves. In their relationship with others these children alternate between aggressive defiance and passive compliance; often the wide variability is demonstrated in the same child within a few minutes.

A. R. was an 11-year-old white boy observed on his third admission. He had been diagnosed schizophrenic at the age of six years. He had episodes of assaultiveness towards his parents. For days at a time he would be mute. At these times he was incontinent of feces, towards which he showed a phobic reaction, glancing at them and then running from them in fear. He would examine his body, then bite his arms and legs. At other times he was quiet, passive, clinging and melting into the arms of adults.

A. N. was a 9-year-old colored boy admitted from school where he was overactive and assaultive. He had auditory hallucinosis, an introjected body and a feeling of being worthless. Following episodes of impulsive assaultiveness, he would apologize, cling and ask for punishment.

Sometimes, as with T. G., such a child asks directly for ego support.

T. G. was a 9-year-old white boy admitted because of truancy, assaultiveness, impulsiveness and distractability. Outbursts of assaultiveness were of such severity that he had to be housed on the disturbed ward. One minute the child would kick and bite; the next minute, cling pleading for help to control himself.

Schizophrenic children who are relatively more successful in defining their egos may try to develop them by imitation. Imitation may be considered an ego function by which the ego tries out many roles and elaborates itself, as exercise is a muscular function by which muscle strength increases. Imitation is considered to be a normal ego function of all people of sufficient ego development. In the non-schizophrenic it is most apparent in the second year of life and in adolescence. In the schizophrenic child it is a prominent feature beyond the expected age. It is socially useful for it is often considered flattering and amusing by people the child depends upon.

B. K. was a 9-year-old white boy admitted because of paranoid ideation, impulsiveness, severe anxiety and distractability in school. On the ward he imitated the mannerisms of one of the doctors and was thrown into a severe anxiety state when the nurses would not follow his orders. He believed that he was being controlled by others. He believed that he controlled the actions of everyone on the ward. When he gave orders that were not obeyed or when ward routine was altered without consulting him, he became anxious, shook and sweated and cried.

The ego function, imitation, should be distinguished from echophraxia and echolalia in which the factor of imitativeness seems prominent. The stereotyped copying of words and movements of surrounding people seems to occur in those schizophrenic children who have not developed ego or body boundaries.

In normal emotional maturation children develop a sense of self by the introjection of parental concepts modified by the child's own ego. In the schizophrenic child with a weak and unreliable ego boundary the introjection occurs in a particularly paradoxical way. The

child has not the ego strength to modify parental concepts. He accepts them preformed. Since they have not been ego-processed, they never seem quite familiar to the child. Sometimes they seem foreign and even inimical. The child has not the ego strength to reject the parental concepts. The introjection is too complete but quite tenuous.

Often the child presents a bizarre picture of a young person with a mental set of an adult. He parrots and acts out concepts he could not be expected to understand. He becomes a small edition of the parent. While the introjection holds, the child may be priggish and "strange" but relatively anxiety-free. The introjection is tenuous just because it is total, unselective. When it fails the child is anxious and to that extent further incapacitated in ego function. If introjection may be compared to swallowing, the schizophrenic child swallows too much food whole and often vomits it back because it is indigestible in the form swallowed.

Manifestations of Over-identification in Different Kinds of Relationships

Mother-patient

A common relationship of schizophrenic child to mother is seen in the case of J. A. J. A. was a 9-year-old white boy admitted because of destructiveness and severe anxiety shown in continuous nagging of his mother and fear of separation from her. For two years before admission he had been failing his school work and had shown so much difficulty in learning that a diagnosis of mental deficiency had been entertained by two competent psychiatrists consulted by the family. For two weeks before admission the child had been placed outside the home by his mother, whose patience had been exhausted by J's continuous whining, nagging and trailing her about the house. It was only when he was separated from his mother that he showed aggressive destructiveness necessitating his hospitalization. At the time of his admission, because of J's pleading, crying and clinging, the mother in a paroxysm of guilt signed his release. After mother and child had been separated for 15 minutes, the mother regained an adult perspective and permitted the child to stay in the hospital. J's entire hospital course, like that of many anxious, over-active schizophrenics, was marked by repeated crises of this sort in which the mother perceived the child's anxiety as a reproach and reacted to it as if it were.

A more uncommon but not rare relationship is seen in the case of K. B. K. was a 3½-year-old white boy of superior intelligence admitted because of insomnia, night terrors and refusal to be separated from his mother. He undertook nothing of his own initiative. He ordered his mother to do everything as his agent. Moreover, he was obsessive-compulsive about how she did things. For example, though he was perfectly able to feed himself, he would not until his mother had fed him the first three mouthfuls, and the feeding had to be done at a certain time of day in a specified manner. When these ritualistic needs were satisfied, then and only then would he feed himself. Rituals permeated all his other daily activities. In treatment play sessions with K and his mother what was remarkable was the two-way nature of the relationship between them. Though the mother verbalized resentment of K's demands, she complied with them, anticipated and fostered them. In the treatment room Mrs. B. identified with K to such an extent that she stimulated neurotic defenses in him so that his diffuse anxiety would be hidden from the therapist. "I don't want even you to know how sick he is. I want him to make a good showing."

Patient with peers

Frequently on the ward a symbiotic relationship is established between two patients. When one of the pair is schizophrenic, over-identification plays a great part in the relationship.

C. B., an 11-year-old white boy, was admitted because of seclusiveness, threatened assault of his mother and scattered talk in which he referred to himself as a great scientist and a President of the United States. On the boys' division of the ward there is always a "king," a boy who leads the rest. Gaining this title is dependent upon true leadership ability, the will to power and wrestling skill. Though C was a strong intelligent boy with a great desire for prestige, he took no part in competing for the title and in fact refused to defend himself when the reigning "king," frightened by C's size, challenged him. C withdrew from all activities until a tense, depressed boy of his own age was admitted. Thereafter they were never apart. The second boy, J. G., was not schizophrenic and had a stronger ego than C's. By a set of extremely unfortunate circumstances, following an accidental act of violence, J. G. had been separated from all companions of his own age for three months and by the time he arrived on the ward he was starved for companionship. He and C. B. immediately got together, planned escapes, played together, initiated group activities together. With the help of his alter-ego C. B. was able to establish a position of relative supremacy on the ward. With this satisfaction his anxiety subsided

and he talked more coherently. When J. G. was discharged, C. B. was unable to maintain his ward position alone and relapsed into a state of anxiety, scattered talk and paranoid ideation.

Doctor-patient

B. U., a 7-year-old white girl, was admitted because of overactivity, aggressiveness, assaultiveness and failure to learn. She clung to or alternatively teased and slapped her physician. She wanted the physician's company continually. She received a course of 20 electric shock treatments. Immediately upon awakening after each treatment she would ask her doctor "Who am I?" She shook her head when told she was B. U. She insisted she was Dr. O. and Dr. O. was B. U. Within a half-hour after treatment she would say the same thing only jokingly, in fun. By afternoon she referred to herself as B. U. and to her doctor as her father disregarding the sex and appearance of the physician. Within a week of completion of her course of electric shock treatment she had established her own identity and other people's even in fun.

Problems Presented by Over-identification in Treatment

These will be discussed under three headings.

1. Uselessness of trying to establish the classical therapeutic relationship.
2. The mother's side of the symbiosis.
3. Acting out.

Uselessness of trying to establish the classical therapeutic relationship

The cardinal principle of psychotherapy is the establishment of a relationship between patient and therapist which relationship is thereafter examined and interpreted by the therapist to the patient. Whether the means of communication be words, gestures, toys or whatever, the principle remains the same. It is a principle not applicable to many schizophrenic children because it is not possible to establish a reliable stable therapeutic relationship to begin with. More often this is so because of the child's difficulty in locating ego boundaries and because of his rigid defenses against the anxiety this generates.

The natural course of schizophrenia is characterized by so-called remissions when the child, driven by anxiety, seeks and finds a pattern of behavior approved by the parents who then call him well. When this happens in treatment the change is sometimes ascribed to the therapist and the parents withdraw the child from treatment convinced that he is cured. When the change is due to a sudden integration of development, such as occurs in the maturation of any child, the improvement may last and the child develop steadily even though periods of additional stress such as starting school or entering adolescence. When the improved behavior is based on over-identification, disappointment is almost certain to follow. Alternating remission and relapse are common. In clinic treatment alternating parental hope and disappointment are problems for the therapist not only because of a natural sympathy with their feelings but because the schizophrenic child who may be overidentified with these adults will reflect their disturbance and add it to his already chaotic sense of self.

The "mother's" side of the symbiosis

Whenever a schizophrenic child is taken on for treatment, at least one adult must be too. This adult is the one who "mothers" the child, whether it is the child's real mother or any substitute adult who supplies the child's bodily needs and teaches him the requirements of the culture. It is from this adult that the child is most apt to derive his sense of self. Henceforth this adult will be referred to as the mother.

On the children's ward of Bellevue Hospital, where many of the children are sent in by the court as neglect cases and where poverty and irresponsibility are probably more common experiences for these families than for families reported by other writers, the parents of schizophrenic children are outstanding as a group for their concern and responsibility for their children. The mother is bewildered by the schizophrenic child's demands and guiltladen because of her inability to respond. After three months of treatment one mother who had a schizophrenic child and a normal child described her feelings: "I can't satisfy him. It isn't just that he wants me around all the time. I can do that. He seems to want my soul and I don't know what to do for him." This is the mother of K. B., the compulsive ritualistic child previously described. For over a year this mother had not had any time to call her own because she had felt compelled by her child's anxiety to stay with him all the time. Though the boy had a room of his own his crying at night kept one or the other parent with him most of the night. It should be noted that parental company did

not comfort him but it comforted the parents. "We did the best we knew how." The point is that the anxiety of the schizophrenic child arouses the anxiety of the responsible adults.

This is seen not only in parents but in ward personnel of proven emotional stability, some of whom have 15 years or more of experience in caring for these children. The anxiety of the schizophrenic child is hard to withstand. Among the student nurses who change ward service every 4 to 8 weeks, an answering anxiety is frequently observed. Before they have had a chance to learn to identify schizophrenics, the new staff members single out the schizophrenic children upon whom to lavish concern and general mothering. They complain to the older staff members that "those children don't belong here. All they need is mothering." To see their bewilderment when they realize that many of these children have good mothering and cannot respond to it amounts to a laboratory demonstration of the complex relationships of the schizophrenic child and his relationships outside the hospital.

The mother responds to the schizophrenic child's anxiety just as the ward personnel do. She lavishes concern and affection upon him. She is bewildered by his failure to respond. She feels defeated and usually looks for the cause in herself rather than in the child. She reads literature on child care and examines her own feelings and thoughts. Like most people, she is not perfect. She selects one of her faults as the probable cause of the difficulty and tries again. With each defeat she repeats the cycle. At some point almost inevitably she will get corrobation from the environment that the fault is hers: A relative, a neighbor, a teacher, or perhaps a police officer will indicate that she is not fulfilling her responsibilities.

This sequence of events is very common and accounts for the second big problem that the schizophrenic child presents in therapy: The relatedness of the mother to the child. This is composed of 1) First an empathic response of anxiety to the child's anxiety; 2) A sense of bewilderment over failure together with a feeling that it must be her fault; 3) A resentment of others outside the mother-child unit who tend to corroborate her unpleasant ideas without helping her.

Acting out

The third problem the schizophrenic child presents in clinic treatment is his greater tendency to act impulsively outside of clinic in a way which makes the environment demand institutionalization.

P. K., an 11-year-old white boy, was referred to clinic by the school because he was inattentive in class and often truanted. His talk was scattered. He whirled. He was preoccupied with processes of growth and how the world was made. He had a close friend, a non-schizophrenic boy of his own age, who gambled and truanted. Under the aegis of this boy, P. K. did likewise. He had a psychotic mother who was hospitalized frequently for manic episodes for which she received shock treatment. After three months' treatment, P. K. had to be hospitalized because in overidentifying with his mother he had reflected an impending psychotic episode in her. For two weeks prior to his admission the mother had been developing the pattern characteristic of her attacks: Paranoid ideation, excitement, flight of activity and threats to leave home. P. K. was enormously concerned with the threat of her loss. He became tenser. He threatened suicide and tried to run away from home. Within a week of his admission the mother was admitted in a state of manic excitement in which she had paranoid ideas about her husband and threatened to leave the home.

Constructive Uses of Over-identification in the Treatment of a Schizophrenic Child;

Further Reports of the Case of K. B.

More classically in the treatment of children, the patient and the parent are seen separately, sometimes by different therapists. Geleerd (7) among others has described the necessity for providing ego support and what amounts to maternal care for schizophrenic children in contrast to what is offered to non-schizophrenic children: interpretation of fantasy material and behavior in the play room. In the case of K. B., already described, it was felt that since K.B.'s ego boundaries seemed to blend with his mother's, to separate him from his mother during treatment would be to damage rather than strengthen his ego. Therefore K and Mrs. B. were seen together in once-a-week interviews for 36 weeks.

The aims of treatment were: 1) To get K identified to himself as himself. 2) To get K to do things alone and enjoy doing so. The means by which this was attempted were: 1) Direct interpretation to the mother of what she was doing with K and on the basis of per-

sonal history she gave, guesses as to why. 2) Encouragement of the mother to do those things she wanted to do for herself and for K which she was afraid to do lest K be disturbed.

K was provided with the equipment for doing the things he liked to do (listen to music, make music, ride a rocking horse) and for doing things he was afraid to do (make a mess with paint, clay or blocks.) At first he would not do anything but melt in his mother's arms. She urged him and forced him to do things he did not want to do. Except for the amenities of saying hello and goodbye, the child was not directly approached. K was as distrustful of the therapist as of all strangers and dealt with her as such: he walked around her at a distance; he was elaborately inattentive when spoken about and glowered when spoken to. At home he repeated what the therapist said to the mother and asked the mother questions about it.

A reliable therapeutic relationship has been established between therapist and child and therapist and mother. Each relationship is dealt with at its own level and it is probably primarily this distinction of level which is useful to both patients.

The child initiated a positive contact by waving at the doctor while looking the other way. Later he would announce that he had something to say, whisper it to his mother and then glower fiercely at the doctor while the message was being delivered. Still later he offered a few direct monosyllables and then a drawing of his friend. The relationship is maintained by acceptance of K's tentative smiles and his proffers of invitations to join play.

In nine months K has accomplished the following: 1) he sleeps through five nights out of seven, 2) he has one friend of his own age, 3) he is able to leave his mother's side to make initial contacts with his peers, 4) he can tolerate changes in household routine, 5) he can play alone in his own house, 6) he can make a mess and make mistakes without undue anxiety.

The mother has accomplished the following: 1) she can leave the child with her own mother to go out for an evening of pleasure with her husband and enjoy herself, 2) she can talk about K to her husband without blaming him for the child's troubles, 3) she is beginning to develop the sense of humor and self-respect that she had before she was overwhelmed by her son's problems.

K is now able to attend nursery school. In the past month the therapeutic situation has been altered by the addition of another mother and child with a similar problem. Gradually a group of four children and mothers will be formed for continuous follow-up.

In the treatment of this patient a pathological situation of over-identification was manipulated in such a way that true ego identification could be started. It is tentatively suggested that similar manipulations might be useful in other situations where over-identification may be one of the roots of a disorder. For example in a classroom group situation where two children are always into mischief it is common to separate them as the quickest way of ending the mischief. A teacher who can manipulate the twosome constructively might foster ego growth for both children as well as end the classroom disruption. In the treatment of schizophrenic children, placement away from an emotionally disturbed parent as in a boarding school or in a foster home is often undertaken for the presumed benefit of the child. However, if the child is better able to relate to or identify with the morbid adult it may be that the satisfaction of being able to relate at all is more valuable than the relatively unusable opportunity of relating to or identifying with healthier adults. It seems possible that manipulation of the already existing parent-child relationships might be more economical. Further investigation of the identification functions of child schizophrenics seems indicated. This is underway in the group sessions being formed around K. B. as described in the preceding paragraph.

BIBLIOGRAPHY

(1) Agoston, Tibor: *Psychoanalyt. Rev., 33:* 1946.
(2) Bender, Lauretta: *Am. J. Orthopsychiat., 22:* No. 2, April 1952.
(3) Bender, Lauretta: *Am. J. Orthopsychiat. 17:* 40, 1947.
(4) Erikson, Erik: *Childhood and Society,* p. 69. New York: W. W. Norton, 1950.

(5) Ibid, p. 77.
(6) Erikson, Erik: *Childhood and Society*, p. 67. New York: W. W. Norton, 1950.
(7) Geleerd, Elizabeth: *Psychoanalytic Study of the Child*, III/IV, 311. New York; International Universities Press, 1949.
(8) Gurevitz, Saul: *Quart. J. Child Behavior, 4:* No. 2, 139-153, April 1952.
(9) Gurevitz, Saul: *Psychoanalysis, I:* No. 1. 62-73, 1952.
(10) Sullivan, Harry S.: *The Interpersonal Theory of Psychiatry*, p. 173. New York: W. W. Norton, 1953.

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PSYCHO-SEXUAL IDENTIFICATION IN THE EPILEPSIES

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While a number of authors (1, 2, 3, 4) have commented on the disturbances of the sexual life of epileptics, and others (5, 6, 7, 8, 9, 10, 11) on the psychodynamics of epilepsy, there are relatively few references to the dynamics of psycho-sexual development in the epilepsies or its relevance to the epileptic attacks. Freud refers to the association of Dostoyvesky's repressed homosexuality with his "affective epilepsy" (5). Cobb (7) mentions the relationship of epileptic attacks to "reversion to homosexual temptation," in one of his cases. In one of the woman patients reported by Heilbrun (8), "sexual intercourse was a beautiful experience only when she was the aggressor or assumed masculine position in the act."

In the two cases of epilepsy reported below —one with both major and minor attacks, and the other with atypical psychomotor attacks— an attempt is made to formulate the psychodynamics with a focus on the psycho-sexual misidentification, which is a fundamental factor in both the cases, though the clinical picture and behavioral manifestations vary considerably.

Case..1. Mr. E. S., a 27-year-old unmarried Jewish man, came to the New York Hospital with the main complaint of "epileptic fits" of about 11 years' duration. These fits were of the classical major and minor types, though the frequency and kind of fits had shown variation over the years. Starting when he was 16 years old, the minor kind had steadily increased in frequency, reaching a peak number of 40 to 50 a day, when he was about 20 years old; after this period, while the minor kind decreased, the major variety came into the picture. At the time of his reporting at the hospital he was having both types of seizures, at the average rate of one every week or two. He had no other bodily complaints except a general state of increased tension which he was in the habit of "working off" by some physical activity, which most often took the form of cooking, house-cleaning and at times, breaking pencils.

Previous health. There was no history of major ailments pertaining to the nervous or other bodily systems, or history of head-injury.

Family history, with reference to the incidence of epilepsy, migraine, or other nervous or mental disease, was negative.

Physical examination at the time of admission revealed him to be about 25 years old, short and stocky in build and ambulatory. Neurological examination was essentially negative and a review of the other systems showed no significant abnormality.

Laboratory investigations. Urine and blood counts and Mazzini's test were essentially normal. Electroencephalogram revealed "severely pathological pattern showing many discharges with a maximal defect in the left frontal and parietal areas."

Psychiatric examination. The patient was neat in appearance and well dressed. He was cooperative, polite, ingratiating and freely communicative. Though the amount of free-floating anxiety was not great he was tense and fidgety during the interviews. Obsessive-compulsive features were in evidence; he brought his diary for help and had entered therein the complete list of events of the day, with particular emphasis on the nature and number of fits he might have had with his own comments regarding their causation. An entry would run more or less as follows: "Had 3 minor fits to-day; must be because I drank too much water yesterday . . .; I must take more vitamin A in my diet." Another item entered with the minutest care and regularity was the nature and number of bowel movements, with comments on the possible relationship between them and his fits. His mood appeared appropriate and he was not depressed; on the contrary, he tended to crack jokes and puns, quite often overdoing them.

He was of above average intelligence, and there was no evidence of gross defects in memory, orientation, perception and judgement.

Personal history. Both his parents were Russian Jews. He was the last of four sons with no sisters. There was an age difference of 8 years between him and the next older brother; "when I came into this world I wasn't really expected, and in addition my mother was very disappointed I wasn't a girl. Anyway she brought me up as a girl and I too enjoyed it obviously, for I used to love cooking, sewing and washing floors, and even today I love cooking different spiced foods".

Father died 4 years before the patient's coming to the hospital, having suffered for a few months from "cancer of the ascending colon." "He worked as a fur-finisher. He used to be easy with all of us; in fact I used to consider him like another of my brothers. I can still remember how he used to kiss me on my lips; the memory of his soft lips, the pores on his skin of the face etc. are all still vivid in my mind" (cried at this point, though quickly controlling himself apologetically).

[1] This study was carried out during the tenure of Post-Doctorate Research Fellowship offered by the Life Insurance Medical Fund.

Mother was "the boss in the house." Though she was not severely punitive, she was strict and fussy about the way things were done: "when I dried dishes, she used to be particular that there was not even a single lint sticking to the dish; when I grew up I used to feel bad that other boys were having a good time, while I had to do my music lessons, but still I would do anything to please her." She died about one year before the patient's coming to the hospital: "this was a terrible blow to me; I cried and cried."

He was apparently on good terms with his brothers during his boyhood days; he remembered how he used to act as a go-between for them, in their heterosexual contacts. "It made me feel good, being associated with them in their love-making." After they all got married however he "began to lose all respect for them, because they were being led by their wives and I had no use for them anymore." Since his mother's death, the relationship had become very strained and at times even hostile." "I don't want to have anything to do with them; all that they are after is my mother's estate set aside for me. I have decided that the best thing to do is to keep aloof, for if things get too bad, I don't know what I'll do."

School and occupational history. He graduated from high school when 17 years old and had been going to night school since, studying for a diploma in Chemical Engineering; "that was one thing my mother wanted me to get, so much." His performance at school had been above average; while he did well during the academic sessions, he felt very tense and diffident during the examinations, and his fits increased in number also. The relationship with the teachers had been satisfactory.

After he finished high school he began working, first as a sales-clerk in a drug store and later as a chemical analyst, which job he had held up to the present. He felt tense whenever he had to work in a competitive situation. In the last place he worked, a few weeks after he was hired, an inexperienced college graduate was also taken. Though the patient was much more experienced in the work and had to instruct the new graduate in all practical details, the latter was paid much more than the patient. When he asked the boss for a raise, he was told his work would be observed for a few weeks and then the question of raise would be decided. Two days later he had a major fit and several minor ones.

Psychosexual history. As indicated earlier, he used to enjoy associating himself with his brothers' sexual activities, even when he was ten years old. At the age of thirteen, he began to indulge in mutual masturbation with a group of four or five boys: "If I didn't masturbate almost daily, I used to get my menstruation—I am sorry, I meant seminal emission." This went on till he was sixteen years old; he then began to wonder "if there was any difference in feeling between masturbation and sexual intercourse." He started to take girls out, but could never get to the point of having intercouse because, "they wouldn't let me; and I didn't know how far I should go." He had his first heterosexual intercourse when he was twenty years old; went in the company of a few boys and at their instigation agreed "to seduce the woman." She was much older, and apparently the more active partner: "it was more a question of her seducing me rather than me seducing her." A few months later he had a similar experience with another woman: "it was all work and little enjoyment for me." He never had any more heterosexual relationships since. He denied any homosexual experience, except for the mutual masturbation referred to.

When he was twenty years old the last of his brothers got married; also around this time he got interested in a girl and was going steadily with her. After a period of courtship and with the active encouragement of his mother, he decided to propose to her; when he did, she agreed—but two days later he withdrew his proposal and "even to this day, I don't know why." He had all along been planning to get married and settle down, but "I can't find a person like my mother; I wonder if I ever will." He had been masturbating with heterosexual fantasies, but rather infrequently.

Social history. His social life had been rather restricted because "most of my friends have got married and I don't want to impose myself on them." Since his mother died he had been living in the same apartment alone, doing the cooking, washing, etc. himself. He had always felt "self-conscious" about his height, and with his epilepsy added to this, he felt very uncomfortable especially in the company of girls. "I am rather irritable and won't take any insults from anybody. The janitor of our building, for example, has been behaving nastily to four or five tenants; I don't feel like seeing him, because if I get into arguments, I don't know what I will do." There was no history of addiction to alcohol, tobacco or other drugs.

Dream and phantasy life. His dreams were simple, with minimal elaboration or complication. For example, he had two dreams on the same night: "In the first one I am having sexual intercourse with an older woman; she is actually seducing me, and I relax; in the second, I am with a girl of my own age, in a group of five or six couples. There is no sexual relationship here except 'the rubbing during dance,.'" Feeling state in the dream: "In the first, I feel very happy; in the second, I am tense."

With little help, he was able to see that the only satisfying heterosexual relationship for him was with older, more active and guiding partners; on the other hand, he felt very insecure and anxious with younger women, and had the situation so maneuvered, say by having five or six couples, that sexual relationship was made impossible.

In another dream, he was at a dance with a blonde-haired boy. This boy showed him the picture of his girl-friend, who looked attractive. A few minutes later, the girl-friend of this boy actually showed up; he (the patient) was very disappointed with her actual looks; she was flat-chested and her

ear-rings were unmatched; his boy friend took one of her ear-rings and our patient teased him about it, singing a rhyme.

Associations. "I used to have a boy friend with blonde hair in my school days; we used to be great chums. . . . I go for girls with big breasts; usually when I go out with girls, I don't go beyond playing with their breasts; I wait for them to give me an O.K. to go farther; but they don't and I am afraid of pressing them; I don't know what I should do . . . . yesterday, our secretary bought two cents worth of pickles for the boy who is working with me; I was teasing him, saying that he was pregnant and was having a craving."

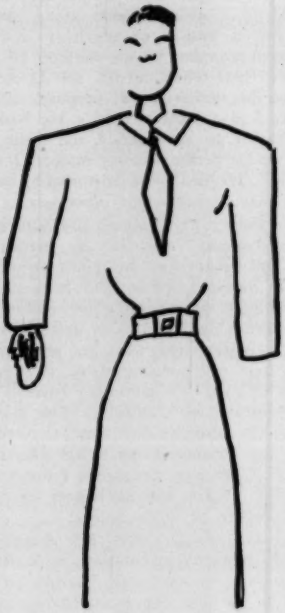

FIG. 1

Human Figure drawings. He started to draw a man's face (fig. 1), then his neck tie, and as he proceeded with his dress he realized that it was more like a woman's skirt. He exclaimed, "Gee! it looks like a woman; what is the matter?" Then he became confused and proceeded to erase the neck-tie in the figure.

When drawing the woman (fig. 2), he started off with the foot and leg and spent nearly five minutes on it, alternately drawing and erasing it; then proceeded upwards, paying great attention to symmetry and proportion of the parts of the body, finally finishing up with the face. The finished drawing represented a small, poorly-organized, feminine looking man and a "phallic" woman; the position of the left leg, in the female drawing, with evidence of marked anxiety as shown by the repeated erasures and thick lines, was significant from the symbolic point of view.

Psychodynamic formulation. This patient's feminine identification and repressed homosexuality, as revealed by his interest in group masturbation, participation in a mediate role in his brother's flirtations during his pre-adolescent years, anxiety and inhibition in heterosexual relationships unless the person is older and phallic, slips of the tongue, dreams and human figure drawings, is determined by an identification with his mother. This again might have various determinants: 1) the fear of rejection as he came at an unexpected period—eight years after the third son—and to add to that, he was also a boy. 2) A "castration anxiety" which is supported by the irrational anxiety and inhibition associated with heterosexual relationships except in a concealed feminine role and with "phallic" women, and by an observation while he was drawing the female figure: he started off with the woman's leg and spent nearly five minutes over it, and the final figure

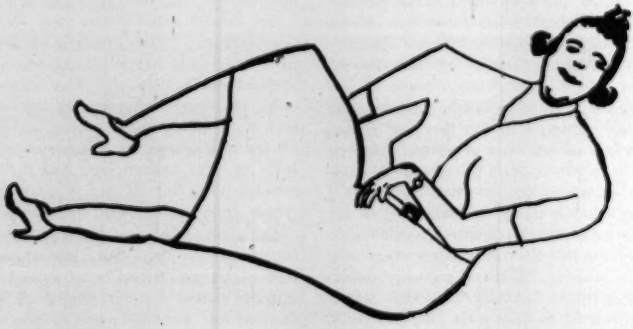

FIG. 2

had an arresting, thick-lined left leg and the female looked "phallic" in more than a symbolic sense. This suggests the mechanism of handling the "castration anxiety" in many foot fetishists (5).

It will be remembered that situations calling for masculine qualities, like heterosexual relationships, or competitive efforts with men of equivalent qualifications, are particularly anxiety-provoking and also bring on the fits. His fits showed up for the first time when he became an "adult," and were at their worst around the age of twenty, when his masculine identification was put to severe test: he had to decide whether he was going to marry the girl he "was going out with" or not. His older brothers, who were acting as "props" for his poorly developed masculine assets and gratified his passive homosexual longings in a concealed way, got married about this time. His mother also started to urge him on for more and more effort in adult participation, and "be like my older brothers." He had always tried to satisfy her. "She was like God to me and I would do anything to please her."

It should be pointed out that the original identification with the mother was predominantly by fear of loss of love and probably by active "castration anxiety," and it was with the "tender" aspect of the mother and hence the feminine orientation in the picture. The aggressive aspect of the mother was repressed and some of it probably bound in the formation of obsessive-compulsive traits as shown by his obsessive diary-keeping and preoccupation with his bowels. That the repressed aggressiveness threatens to "return" at times, and is anxiety-producing to the patient, is shown by his avoiding the "too bossy" janitor, whom he might "even kill," and also his avoiding his brothers for more or less the same reason. Repression of aggressiveness means, to the patient, repression of even normal assertiveness and competitive effort with other men, so that the situation calling for a show of these qualities is also anxiety-provoking, and in most cases brings on the fits, too. Under these circumstances, the fits may be construed to mean 1) an escape from a difficult situation (11), 2) an outlet for his aggressiveness and self-punishment for the same, and 3) gratification for passive, receptive longings.

Case 2. Mr. J. L., a 37-year-old Catholic married man, came to the New York Hospital with the main complaint of "weakness, nervousness and trouble with the prostate gland." The real problems he came to the clinic for, we gathered later, were 1) Impotence, and 2) Outbursts of severe assaultiveness coming on after the slightest provocation, with loss of consciousness and amnesia for the assault and associated events. Both these main complaints were of about 19 years' duration.

The impotence was not absolute. Though he never had an erection when he wanted it, it was possible to produce one by "fooling around with the woman's leg"—i.e., by rubbing his penis against the woman's leg for some time before the actual intercourse. The erection thus obtained was painful and the ejaculation was incomplete. He had tried to have intercourse with different women, but had always failed. He could never get even this kind of erection with women who were total strangers to him.

The impulsive outbursts of assaultiveness were usually provoked by someone trying to argue with him. They had always been directed against older men, never against women or men younger than the patient. He did not remember the actual assault, but "passed out." The last time this happened was in a bar, where he was working as a bartender; he told a customer, an older man, that he was drinking too much, and the next thing he remembered was the customer lying on the floor with bruises over his body, and three men holding the patient. He learned later that he had attacked the other man, passed out subsequently, becoming restless while recovering. There was no history of convulsions or loss of consciousness unassociated with the assaultive outbursts.

Family history, with reference to the incidence of epilepsy, migraine or other nervous or mental disease, was negative.

Physical examination. He was an asthenic white man, 37 years old and ambulatory. Neurological examination was essentially negative and a review of other systems revealed no significant abnormality.

Laboratory investigations. All relevant tests were negative except electroencephalogram, which revealed "mildly pathological pattern, because of alpha spiking," especially in the left temporal area.

Psychiatric examination. The patient was a neat, well-dressed man, communicative in a restricted way, most of his answers being short and simple. Attempts to get him to elaborate any further were unsuccessful. He appeared tense and on guard, and putting him on the couch made him very uncomfortable, as he started to complain of pounding of his heart and pressure on the back of his head, which subsided on sitting up. He also tended to avoid the examiner's gaze. While his mood was not one of depression or bizarreness, there was a rigid monotony about it.

He appeared to be of average intelligence and there were no gross defects in memory, orientation or perception: He did however give evidence of mild ideas of reference: "Yes, sometimes I get the feeling that people are looking at me and talking about me."

Personal history. The patient was born of American Catholic parents, the second of three sons. Father was working as a superintendent of buildings, drank heavily, and was very "cruel" to the mother. The patient hated him right from the beginning, when he was thirteen years old challenged him to a fight, but was thrashed badly. He died, when the patient was eighteen years old, of "hardening of the arteries." Mother was fifty-seven years old, well and living with her second husband. The patient had always "respected" his mother. He didn't like his step-father because "he didn't treat mother right."

School and occupational history. He had finished eighth grade and then worked in many unskilled jobs; spent two and a half years in the army as a truck driver and got an honorable discharge in 1945. He held many short-time jobs after that and at the time of admission to the clinic was working as a garage mechanic and also superintendent of a building.

Psychosexual history. When he was twelve years old, he took a girl out along with another boy and his date. Both the boys tried to have intercourse, but while the other boy succeeded, the patient failed. He masturbated quite a lot during this period and subsequently: "It was more like a game; four of us used to get together and do it to find out who could hold on longest". He denied indulging in overt homosexual or other perverse sexual activities.

At one time he used to get an erection at the mere sight of a woman's leg. As indicated earlier, one of the pre-conditions for a heterosexual intercourse was "fooling around" with the woman's leg. He had his first heterosexual intercourse when he was eighteen years old. This was soon after his father's death from "hardening of the arteries." During the act of copulation, the girl pressed firmly on the back of his head; he heard a sudden snapping sound, "it was like a blood vessel rupturing," and he could not carry on with the coitus. He attributed his impotence to this experience.

When he was nineteen years old, he married a woman ten years older, who had had many operations of the "womb"; "her womb and half her inside had been taken out." He denied any knowledge of this at the time of marriage; had the marriage annulled when he was twenty-nine years old, after he saw her in bed with another woman. He got married again when thirty-five years old to a girl eighteen years of age. It was at her instigation that he decided to come for treatment for his impotence as she was keen on having children. He had tried to have sexual intercourse with different women but without any success.

Early memories. When he was seven or eight years old, he saw for the first time "a middle aged woman's private parts and got scared." Soon after he started to have a phantasy: he "used to think that when a woman had intercourse, the man's part may get left behind inside the woman and that it is taken out by operation." When he was about twelve years old, he once saw a man being kicked on the face by another man, and his face was all covered up with blood. On another occasion he saw a boy being run over by a car; "it was all mashed up like pieces of raw meat."

Social history. He had only a few friends and felt uncomfortable in the company of men, especially if they stared at him or followed him. "The man who had an affair with my first wife was a good friend of mine. I don't trust no man no more."

Habits. He drank and smoked moderately.

Psychodynamic formulation. This man's impotence is most probably the result of "castration anxiety," as shown so patently by his early memories, which evidently may be "screen memories." His pre-condition of heterosexual intercourse, namely "fooling around with the woman's leg" is probably a reassurance-seeking measure to allay his "castration anxiety" (5). As for his assultive outbursts, it must be remembered that they are directed against men older than himself and that they are followed by loss of consciousness with amnesia for the outburst and associated events. It should also be remembered that the patient was consistently hostile towards his father, right from the time he could remember any events at all, and this was manifest towards his step-father also.

One way of looking at this pattern is that he turned to father as the object of his erotic feelings, identifying himself with the "castrating" mother; and his hostility to father and his symbolic substitutes was a reaction formation against such feelings. His anxiety when made to lie down on the couch and when followed by another man also suggest a repressed homosexual component. The loss of consciousness itself may be seen as a mechanism to keep out of awareness the intensity of his own aggression, and symbolically seen as a self-inflicted form of death, to expiate for his aggression.

Discussion

These two cases are similar in that they both show difficulties in normal heterosexual intercourse, and also manifest a fundamental "de-

tensive" identification which varies in each case. In spite of the difference in type of epileptic attacks, the situations precipitating them are also similar. These are usually of the kind that intensify the conflict associated with psychosexual misidentification; at a deeper level they may be construed as reactivating the conflicts relative to their feelings towards the fathers and father-surrogates.

Their behavioral differences can be understood if one considers the degree of acceptance or denial of this identification, and also the degree of tolerance of their own aggressiveness.

Summary

A psychodynamic study of two cases of epilepsy is made, with the focus on their psychosexual identification and its possible relevance to the epileptic attacks.

My thanks are due to Drs. Harold G. Wolff, Thomas A. Rennie and Oskar Diethelm for their critical comments.

Bibliography

(1) Aring, Charles D.: The role of emotion in the causation of epilepsy. In: *Epilepsy*, Proceedings of the Association held jointly with the International League against Epilepsy, 1946. Baltimore: The Williams & Wilkins Co., 1947.

(2) Bloch, I.: *The Sexual Life of Our Time.* New York: New York Allied Book Co., 1928.

(3) Cobb, S.: Psychiatric approach to the treatment of epilepsy. *Am. Psychol., 96:* 1009, 1940.

(4) Freud, S.: *Dostoyevsky and Parricide.* Coll. Papers, Vol. 5. London: The Hogarth Press, 1950.

(5) Fenichel, Otto: *The Psychoanalytical Theory of Neurosis.* New York: W. W. Norton, 1945.

(6) Greenson, Ralph R.: On Genuine Epilepsy. Psa. Quart. 13: 139, 1944.

(7) Heilbrunn, Psychodynamic Aspects of Epilepsy. Psa. Quart. 19: 145, 1950.

(8) Kardiner, A. The Bio-Analysis of the Epileptic Reaction. *Psychiatric Quart.* 1: 375, 1932.

(9) Krafft-Ebbing, R. V.: *Psychopathia Sexualis:* Chicago: Login Bros., 1931.

(10) Maeder, A.: Sexualitat und Epilepsia. *Jahrb. Psychoanal. Psychopath. Forsch, 1:* 119, 1909.

(11) Mittelman, Bela: Psychopathology of epilepsy. In. *Epilepsy* (edited by Paul H. Hoch and Robert P. Knight. New York: Grune and Stratton, 1947.

(12) Stekel, W.: *Impotence In the Male, Vol. 2:* New York: Liveright Publishing Corp., 1939.

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CONVULSIONS IN CHILDHOOD AND EPILEPSY

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There are two classical forms of approach to study convulsions in childhood: one, which we may call prospective, sees the infantile accident as a prognostic possibility and, for a child who has had convulsion tries to answer the question: Will he be an epileptic in the future? This is the point of view of some pediatricians (Marfan, Thom, Still, Pigott, Weingrow, Fitch, Peterman, Marchand, Debré, Levy, Babbonneix, Hueyer, Dublineau, Gareiso and Escardó). The other approach, which we may call retrospective, considers the infantile convulsion as a past event, apart from the present epilepsy, and tries to find its significance in the etiology of the disease. This is the angle of the neurologist and the psychiatrist, especially the one who studies epilepsy in the adult (Herpin, Beau, Dufour, Muskens, Abadie, Lennox, Gowers, Feré, Patrick, Levy, etc.) The present investigation follows the latter orientation and was done with the purpose of finding the frequency of childhood convulsions in groups of adults, epileptic and non-epileptic patients, trying to find the relationship of the childhood convulsive fit to chronic epilepsy.

By "infantile convulsion" we do not mean all the nervous fits of childhood—epileptic, spasmophilic, psychogenic, etc.—but only those which are different from chronic epilepsy. Abadie called these fits "eclamptic infantile syndrome." There are isolated or grouped paroxyms, completely independent of a chronic convulsive process and known to the author only through the anamnesis. Generally they are seen in one day or in two or three consecutive days, exceptionally they last two or three months. In epileptic patients we have established a minimal interval between the early crisis and the begining of epilepsy proper of seven years for infancy and ten years for childhood.

The main purpose of this work is not clinical but etiological and we have tried to answer two questions: 1) what differences and similarities are there between infantile convulsions and epilepsy? and 2) is there any relationship between the clinical aspects and the causes of infantile convulsions and epilepsy?

MATERIAL AND METHODS

The case material is formed by two groups, one of 1221 ambulatory epileptic patients, of whom 241 had information of infantile convulsions, and the other, of 512 nonepileptic subjects studied as controls to the former.

The patients have been divided in several subgroups, as seen in the tables.

The retrospective character of our investigation renders difficult the reconstruction and fixation of the accident. The patient, for obvious reasons, cannot give this information and the relatives sometimes do not remember details. Therefore it is not always possible to know the precise clinical pattern of the seizures, but for the goals of this investigation the diagnosis of the clinical variety is less important than the unequivocal data on the existence or absence of the infantile convulsions.

We have divided the results of our investigation into three sections: the first and second deal with the frequency of infantile convulsions in the epileptic states and in nonepileptics and the third section with our results.

INFANTILE CONVULSIONS IN EPILEPTIC PATIENTS

The results can be seen in tables 1 and 2. Table 1 corresponds to epileptics who had a convulsive fit in childhood up to ten years old. Table 2 shows epileptics who had an infantile fit before 5 years of age.

The frequency of childhood convulsions in epileptics varies from 25 per cent in table 2 to 27 per cent in table 1. The reciprocal relationship between cases with or without the "eclamptic syndrome" is maintained.

If we examine the subgroups we may see: 1) that the relative frequency of early convulsions is maximal in females, in essential epileptics, in the severe convulsive processes

TABLE 1.—CHILDHOOD CONVULSIONS IN EPILEPTIC PATIENTS

	With Eclampsia		*Without Eclampsia*		
	No.	*Per cent*	*No.*	*Per cent*	*Total*
Sex					
Men	36	26.08	102	73.91	138
Women	32	31.06	71	68.93	103
Total	68	27.21	173	72.78	241
Native region					
Coast	57	35.40	104	64.59	161
Andes	10	14.92	57	85.07	67
Total	67	29.38	161	70.61	228
Clinical variety					
Essentials	31	34.83	58	65.16	89
Symptomatics	14	20.58	54	79.41	68
Cryptogenetics	23	27.71	60	72.28	83
Total	68	28.33	172	71.66	240
Clinical variety					
Moderate	5	25.00	15	75.00	20
Medium	22	25.58	64	74.41	86
Severe	26	32.91	53	67.08	79
Total	53	28.64	132	71.35	185

TABLE 2.—CHILDHOOD CONVULSIONS IN EPILEPTICS*

	With Eclampsia		*Without Eclampsia*		*Total*
	No.	*Per cent*	*No.*	*Per cent*	
Sex					
Men	34	25.00	102	75.00	136
Women	26	26.70	71	73.29	97
Total	60	25.75	173	74.25	233
Native region					
Coast	52	33.33	104	66,66	156
Andes	8	12.30	57	87.69	65
Total	60	27.14	161	72.85	221
Clinical variety					
Essentials	26	30.95	58	69.04	84
Symptomatics	14	20.58	54	79.41	68
Cryptogenetics	21	25.92	60	74.07	81
Total	61	26.18	172	73.81	233
Clinical variety					
Moderate	4	21.05	15	78.94	19
Medium	20	23.10	64	76.89	84
Severe	21	28.37	53	71.62	74
Total	45	25.42	132	74.57	177

* Comprising only those who had eclampsia from 0 to 5 years of age.

and in the people from the coast of Peru as compared with people from the mountains, and 2) that these relationships are of the same degree in both tables; therefore the "eclamptic syndrome" after five years of age is of the same nature as the one which begins earlier.

FREQUENCY OF INFANTILE CONVULSIONS IN NON-EPILEPTIC SUBJECTS

The results may be seen in tables 3 and 4. The first one shows the frequency of the "eclamptic syndrome" in people who are now healthy and in other patients who have several non-epileptic diseases. We have elimin-

TABLE 3.—CHILDHOOD IN NON-EPILEPTIC SUBJECTS

	With Eclampsia		*Without Eclampsia*		*Total*
	No.	*Per cent*	*No.*	*Per cent*	
Neurotic cases	5	14.28	30	85.71	35
Psychotic cases	1	7.69	12	92.30	13
Oligophrenia cases	—	—	2	100.00	2
Total mental disease	6	12.00	44	88.00	50
Neurological cases	3	25.00	9	75.00	12
Total neuropsychiatric cases	9	14.51	53	85.48	62
Medical cases	10	10.41	86	89.58	96
Surgical cases	2	5.71	33	94.28	35
Organic cases without definite diagnosis	23	24.73	70	75.26	93
Total organic disease	35	15.62	189	84.37	224
Total organic disease including neurological	38	16.10	198	83.89	236
Total	50	9.76	462	90.23	512
Total eclamptics up to five years old	33	6.66	462	93.33	495

TABLE 4.—FREQUENCY OF CHILDHOOD CONVULSIONS IN THE SUBGROUPS OF NON-EPILEPTIC PATIENTS

	With Eclampsia		*Without Eclampsia*		*Total*
	No.	*Per cent*	*No.*	*Per cent*	
Sex					
Men	20	6.07	309	93.92	329
Women	28	15.90	148	84.09	176
Total	48	9.50	457	90.49	505
Native region					
Coast	31	12.75	212	87.24	243
Andes	7	3.28	206	96.71	213
Jungle	3	9.37	29	90.62	32
Total	41	8.40	447	91.59	488
Morbid disposition					
Susceptible organic disease	23	31.50	50	68.49	73
No susceptible organic disease	21	5.53	359	94.46	380
Susceptible mental disease	28	36.36	49	63.63	77
No susceptible mental disease	17	4.53	358	95.46	375
Total	89	9.83	816	90.16	905

ated all patients who had chronic convulsive fits, even the psychogenic cases.

Table 3 shows that 9 per cent of the non-convulsive patients have had the "eclamptic syndrome" and 6 per cent had convulsions only up to five years of age. These figures are five times less that what has been found for the epileptic cases.

Table 3 also shows that the highest percentage of non-epileptic infantile fits corresponds to neurological patients (25 per cent) and the lowest percentage to the patients now healthy (3 per cent). The cases from the Departments of Internal Medicine and Surgery have had infantile convulsions in 15 per cent of them, almost the same proportion as the cases with mental diseases (12 per cent).

Table 4 shows that the "eclamptic syndrome" is more prevalent in women, in people born on the coast of Peru and in the jungle regions of the same country. These facts are similar to what was found in the epileptic group.

Finally, the frequency of the "infantile eclamptic syndrome" is greater in people prone to organic or mental disease compared with steady and healthy individuals.

Discussion

Our findings are similar to what has been found in other countries, as may be seen in tables 5 and 6. These similar results would mean that the incidence of infantile convulsions is due to the same causes. The statistical differences in table 5, concerning epileptics, may be attributed to differences in the people who study fits in children and adults. In the latter the epileptic infantile convulsions are systematically eliminated and furthermore only a part of the "eclamptic" children reach adulthood because many die in infancy or childhood.

TABLE 5.—Frequency of Childhood Convulsions in Non-Epileptic Patients According to Various Authors

Author	*Per Cent*
Investigation in adults	
Sal y Rosas	25.00
Beau	5.00
Bullard & Townsend	16.00
Dufour	27.00
Feré	33.00
Gowers	12.50
Herpin	7.00
Lennox	20.00
Muskens	25.00
Patrick & Levy	19.00
Investigation in children	
Burr	39.00
Gareiso & Escardó	30.00
Heuyer & Dublineau	61.00
P. Marie	75.00
Osler	40.00
Still	42.00
Thom	40.00
Unknown material	
Collin*	64.00
Frinch†	46.00
Monad	44.00
Labourdette & Debort†	56.00
Michea & Berger*	20.00
Longchampt	59.00

† Quoted by Marchand & Ajuriaguerra.
* Quoted by Gareiso & Escardó.

TABLE 6.—Frequency of Childhood Convulsions in Non-Epileptic Patients According to Various Authors

Author	*Per Cent*
Investigation in adults	
Sal y Rosas	6.60
Camargo	7.00
Lennox	5.00
Patrick & Levy	4.20
Investigation in children	
Bullard & Townsen	10.00
Thom	6.70
Walton & Carter	10.00

There is a major frequency of infantile convulsions in the epileptics compared with the non-epileptics. This fact has been observed frequently by other authors.

We have found also that there is a relationship between the frequency of infantile convulsions and the quality and intensity of the epileptic process, as seen in the clinical pattern and in the severity of the epileptic picture. This fact has been investigated and described for the first time in the present work.

Comparing the seizures in epileptics and in non epileptics (tables 2 and 3) we may see that 1 in 20 of the latter had childhood convulsions, while in the former the ratio was 1 in 4. This means: 1) that the central nervous system of the epileptic is from early childhood much more sensitive to convulsive factors than the brain of the non-epileptics, and 2) that the "eclamptic syndrome" has something in common with epilepsy from the etiological point of view, probably on a constitutional basis. This hypothesis is substantiated by the frequency of infantile seizures in essential epilepsy as contrasted with other forms of epilepsy (table 1). Essential epilepsy seems to be more related to endogenous factors, and therefore the genuine epileptic patient is more prone to react with seizures on most diverse stimuli, beginning with early childhood.

The maximal tendency to seizures of the essential epileptic has been discussed by Foerster, Kehrer, Mauz, Stauder, Krestchmer, Bonhofer. Gastaut found that the threshold of

"reactivity"—that is to say the minimal quantity of a stimulus necessary to produce epileptic signs in the EEG—is lower in genuine epileptics and gradually higher in the symptomatic cases and other human types. We found similar facts in 1938, when we established that the convulsive threshold—minimal convulsive dose of metrazol—is always lower in essential than in symptomatic epilepsy.

We also found that there is a higher number of infantile convulsions among the "severe" epileptics, that is to say in the cases of severe or malignant course. This observation may be explained by an early damage to the brain by the convulsions in infancy or by a greater degree of convulsive disposition, which may cause a predisposition to infantile convulsions and a greater intensity of the epileptic process.

The general conclusion to be drawn from what has been said is that infantile convulsions are a sign of a higher convulsive disposition, whose nature does not seem fundamentally different from essential epilepsy. This conclusion is not far from the results of the investigations of Hasse, Henoch, Franck, Pierret, Trousseau, Germain, Sea, Joffroy, Dufour, Fere, Gelineau, Gestaut, Cruchet, Longchampt and Abadie.

Among epileptics and non-epileptics we have found a greater percentage of infantile convulsions in patients from the warm zones of the coast of Peru than in patients from the cold zones of the Andes. This observation probably means that the inhabitant of the coast has a greater convulsive disposition than the one from the mountains. We have described this phenomenon in several studies of epilepsy in Peru.

From table 3 we can learn that in non-epileptic patients the greatest incidence of infantile convulsions is seen in the neurological cases, all of which had meningocerebral lesions. This finding may signify that infantile convulsions makes the brain less resistant to the various noxae.

Twelve per cent of our patients with a history of infantile seizures were suffering from mental disease. All of them were neurotics, with the exception of one who was a schizophrenic. We did not find infantile convulsions in our few cases of oligophrenia, whereas in other reports it is a common finding (Thom, Patrick, Levin, Marchand, Ajuriagurra, Thiemich, Birk, Bullard and Townsend). We believe that this apparent discrepancy is due to the small number of mental defectives in our material.

We would like to emphasize that infantile convulsions are more frequent in individuals who are weak from the organic or mental point of view. From table 4 we may see that patients who are prone to infectious diseases or to psychoneurotic reactions had frequent convulsions in childhood. This would mean that seizures in early life may determine some mental or organic vulnerability or that they occur in individuals of defective somatic or psychological constitution.

We think that spasmophilia, which comprises tetany and laryngospasm, is a syndrome of the group whose center is epilepsy. From the clinical or biochemical points of view we think that there are no precise signs to differentiate tetanic convulsions from atypical crisis of epilepsy. Probably the same is true for "spasmus nutans" and other infantile atypical crisis of a strong psychological content as the "sobbing spasm" (spasme du sanglot) of French authors.

With reference to psychogenic convulsive crisis we do not think that they can be considered as a completely different entity from the biological phenomena of convulsions. We are inclined to assume that seizures, psychogenic or somatogenic, occur always in subjects of a specific disposition to convulsions. Without this constitutional makeup the organic or psychological stimulus would produce other symptoms but not convulsive attacks. The person who has a heightened disposition for convulsions reacts to excitation of his brain with convulsive crisis, typically or atypically epileptic, depending on the more or less typical character of the reaction, episode or epileptic process, according to the penetration power of the hereditary influence and the nature and intensity of the stimulus.

Epileptic attacks precipitated by psychological stimuli are probably due to the emotion only in the beginning of its mechanism. The emotion would produce biochemical changes capable of exciting the brain and precipitate the convulsion. The rest is produced in the nervous elements in an autonomic way, in-

dependently from the emotion. In the hysterical seizures there is a degradation of this mechanism and production of a great variety of convulsive attacks, from minimal movements to automatic hyperkynetic seizures with complete loss of consciousness and a succession of convulsive and neurovegetative symptoms—in the epileptic pattern, but completely psychogenic.

In previous work on the convulsive threshold, the latent time, and the duration of the seizure produced by metrazol in several groups of patients, I found that these elements of the experimental convulsive reaction offer statistically a peculiar gradation from complete akinetic responses (represented by non-epileptics) to confirmed epilepsy. The patients with hysterical reactions showed an intermediate threshold between non-convulsive patients and epileptics. An investigation in convulsive hysterics and nonconvulsive patients showed that the latter were deviated to the akinetic cases and the convulsive hysterics to epilepsy.

The general conclusion from the above observations is that although from the clinical point of view infantile convulsions and chronic epilepsy are different and independent conditions, from the etiological point of view these conditions are ostensibly and repeatedly linked. I do not want to refer to other authors (Lasegue, Pierre Marie, Binswanger, etc.) who believe that the precoccious eclamptic syndrome would be a predisposing cause of epilepsy. I think that there is a deeper relationship, which is common to both conditions and which is seen in two concrete facts: 1) the higher frequency of the eclamptic syndrome in the past history of epileptic patients as compared with non-epileptics, and 2) especially the correlation found in the present investigation, between the intensity of the actual epileptic process and the frequency of fits in the past history. Furthermore, it has been found that about 50 per cent of the cases of childhood convulsions develop epilepsy later on. (Thom, Still, Osler, Burr).

The linkage between the eclamptic syndrome and epilepsy seems to be of a dispositional order, whose roots are lost in the complex mechanisms of constitution and heredity. To us, infantile convulsions and epilepsy are aspects or particular forms of expression of a fundamental biological reality, which can be called convulsive disposition, ictal constitution or simply ictaffinity.

Summary

An investigation has been carried out on the frequency of childhood convulsions of the eclamptic type (acute episode, independent from an epileptic process) in several adult groups of epileptic and non epileptic patients with the following results:

The greater frequency of childhood convulsions in the past history of epileptics is confirmed.

The original facts of this investigation are:

The greater frequency of childhood eclamptic syndrome in essential epilepsy and in the severe or malignant cases of epilepsy, showing that the tendency to early seizures is greater as the convulsive phenomenon is graver.

Among the non-epileptic patients the maximal frequency corresponds to those patients who have organic brain disease and the low frequency to mental patients. This last result seems to indicate that childhood convulsions — a similar phenomenon to epilepsy—does not favor the presence of other mental processes apart from essential epilepsy, which tends recently to be considered as an endogenous psychosis.

In patients susceptible to organic and mental diseases the frequency of the eclamptic syndrome is greater than in the resistant cases.

Childhood convulsions are more frequent in epileptic patients and in non-epileptics born on the coast of Peru, which is a warm and moist climate, as compared with the inhabitants of the cold regions of the Andes. This observation confirms previous work of the author which seemed to show that the tropical climate favors the appearance of convulsions.

The facts described are discussed and a hypothesis is advanced. There would be an etiological link between childhood convulsions and chronic epilepsy, of a dispositional order, and both would be only particular aspects of a fundamental biological reality: ictaffinity or the tendency to develop convulsions.

REFERENCES

(1) Abadie, J.: Epilepsie et convulsions infantiles. *Proc. Int. Cong. Neurol.*, London, 1935.

(2) Babonneix, L.: Les convulsions infantiles. Etude clinique et thérapeutique. *Paris M. 45:* 362-9, 1937.

(3) Baptista, V.: Epilepsie na crianca. *Arqu. Ass. Psicop. S. Paulo. 6:* 285, 1941.

(4) Camargo, O.: O problema da epilepsia encarada pelas escolas norteamericanas. *Arquiv. Brasil. de Hig. Mental, 18:* 59, 1947.

(5) Cremieux, A.: Les crises nerveuses de l'enfant. in *Les Epilepsies*, H. Roger & colab. Paris: Kulammanion. 1950.

(6) Debre, M. R., and Levy, F.: Les convulsions essentielles du nourrison et leur pronostic. *Gaz. Hopitaux, 105:* 385, 1932.

(7) Fere, Ch.: *Les épilepsies et les épileptiques.* Paris: Alcau, 1890.

(8) Gareiso, A., and Escardo, F.: Nota sobre el pronostico de las convulsiones infantiles. *Prensa Méd. Arg., 23:* 2660, 1936.

(9) Gastaut, H.: L'électroencephalographie au cours de l'épilepsie, in *Les Epilepsies*, H. Roger, et al. Paris: Kulammonion, 1950.

(10) Hartenberg, P.: Convulsions infantiles. Paris: *Presse méd., 42:* 895, 1934.

(11) Herpin, Th.: *Du pronostic et traitement curatif de l'épilepsie.* Paris, 1852.

(12) Lennox, G.: Ciencia y convulsiones. *El Ateneo*, Bs. Aires, 1948.

(13) Lesne, E., and Turpin, R.: Spasmophilie. *Presse méd., 34:* 1, 1926.

(14) Lhote, H.: *Etiologie de l'épilepsie dite essentielle.* Paris: J. B. Baillière, 1900.

(15) Marchand, L.: De l'épilepsie infantile. *Presse méd., 33:* 1142, 1925.

(16) Marchand, L., and de Ajuriaguerra, J.: Convulsions infantiles et épilepsie. in *Epilepsies.* Paris: Desclée, 1948.

(17) Marfan, B.: Les convulsions dans la premiere enfance. *Presse méd., 64:* 633, 1921.

(18) Mauz, F.: *La predisposición a los ataques convulsivos. Madrid:* Morata, 1942.

(19) Patrick, H. T., and Levy, D.: Early convulsions in epileptics and in others. *J. A. M. A., 82:* 375, 1924.

(20) Peterman, M. G.: Convulsions in childhood. *J. A. M. A., 97:* 546, 1932.

(21) Peterman, M. G.: Convulsions in childhood. *J. A. M. A., 102:* 1729, 1934.

(22) Sal y Rosas, F.: El cardiazol en la epilepsia. *Rev. Psiq. Criminol. Bs. Aires, 4:* 709, 1939.

(23) Sal y Rosas, F.: Contribución experimental a la patogenia de la epilepsia y de la histeria. Thesis D. M. *Rev. Neuro-Psiquiatría, 5:* 450, 1942.

(24) Sal y Rosas, F.: El dintel de convulsión en la epilepsia y en la histeria. *Rev. Psiq. Criminol. Bs. Aires, 8:* 433, 1943.

(25) Sal y Rosas, F.: Epilepsia experimental y epilepsia enfemedad en función del medio geográfico. *Rev. Neuro-Psiquiat, 8:* 147, 1945.

(26) Sal y Rosas, F.: Obserbaciones acerca de la edad de comienzo de la epilepsia. *Rev. Neuro-Psiquiat., 11:* 171, 1948.

(27) Sal y Rosas, F.: Formas del primer acceso epiléptico. *Rep. Soc. Neuro-Psiquiat. y Med. Legal*, 1951.

(28) Sal y Rosas, F.: Estado orgánico y psíquico del individuo en el comienzo de la epilepsia. *Rev. Neuro-Psiquiat., 14:* 211, 1951.

(29) Sal y Rosas, F.: Probable influencia de las estaciones del año en el comienzo de la epilepsia. *Rev. Neuro-Psiquiat., 15:* 66, 1952.

(30) Sal y Rosas, F.: El comienzo de la epilepsia en las distintas horas del día. *Rep. Soc. Neuro-Psiquiat. y Med. Legal*, 1951.

(31) Sal y Rosas, F.: Observaciones acerca de la influencia de los climas tipicos del Perú en el comienzo de la epilepsia. *Rep. Soc. Neuro-Psiquiat. y Med. Legal*, 1952.

(32) Thom, D.: Infantile convulsions: their frequency and importance. *Am. J. Psychiat., 83:* 613, 1927.

(33) Thom, D.: Convulsions of early life and their relation to the chronic convulsive disorders and mental defect. *Am. J. Psychiat., 98:* 574, 1942.

CHANGES IN THEMATIC APPERCEPTION TEST STORIES PARALLELING CHANGES IN CLINICAL STATUS OF SCHIZOPHRENIC PATIENTS [1]

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Since the TAT "serves to force the subject to deal in his own way with certain classical human situations" (15), it may be used to provide data concerning a variety of clinically important vectors, including the subject's conception of the environment, his relation to it, emotional responsiveness, emotional control (9), relationship to others, and defense against anxieties (23). In this paper we report the relationship between changes in TAT stories and changes in clinical status of acute schizophrenic patients during their residence at Boston Psychopathic Hospital. Three primary areas are considered, (a) interpersonal relations, (b) attitude towards the world, and (c) expression and control of emotions—with particular emphasis on whether or not "withdrawal" in these areas is increased or decreased during the course of hospitalization.

The TAT productions are complex. In order to use them in research the conceptual framework of the study determines to a large extent the treatment of the data (9). It is therefore necessary to present our criteria for evaulation of changes in TAT stories in some detail.

CRITERIA FOR EVALUATING CHANGES IN TAT STORIES

Since the patient's own needs and experiences determine his perceptions, the criteria for evaluating changes in the three areas of "withdrawal" cannot be absolute or even highly specific. In order to have elicited specific data for each story it would have been necessary to modify the test instructions radically, of the test. To avoid this the TAT was administered in the usual fashion, with instructions to tell what was happening in the pictures, the events leading up to the depicted situation, the outcome, and what the characters were thinking and feeling. Questions by the examiner were kept at a minimum.

In order to determine whether changes were present or absent, stories given at different times by the same subject to the same card were read together, in the "horizontal" approach described by Henry (8). The second story was compared with the first story, the rater indicating whether the patient showed "positive change" or "no change." A rating of "positive change" was made if there was evidence of less withdrawal, or healthier responses, on the second story than on the first. The clinical significance of the dynamics in the story was not the concern of the rater. He was required to judge whether the patient showed positive changes in the three areas of withdrawal and not whether the stories indicated mental health or illness or areas of significant conflicts.

The instructions for making judgments of change or no change in the direction of less withdrawal were general, and are described below. Too many examples would be required to cover all the situations in which perception of, and attitudes toward, others, the world and emotions were communicated in the TAT stories.

I. Positive changes in interpersonal relations

A. An increase in ability to accept people with more definite roles and with less ambiguity or generality was regarded as evidence of an increase in feelings of closeness to others and hence less withdrawal from them.

[1] This study was sponsored by the United States Public Health Service in the form of a fellowship grant to the senior author.

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If the second story showed clearer descriptions of the people and their identity, or indication of decrease in psychological distance between the people perceived, the change was regarded as positive.

B. Describing events of the second story in the more immediate present than the first story was regarded as indicative of a lessening of the barrier between the patient and others. A change to a "me—here—now" attitude from a "they—there—then" one suggested a decrease in the psychological barrier. Change to present tense when past tense had been used previously was also evidence of increase in closeness to other people. Specifically, this change can be exemplified by a statement on a second story such as "this is a mid-west farmer" while on the first story the patient said "this is in biblical times."

C. Greater closeness to others was also indicated by change in the identity of the figures. Identifying two women as "mother and daughter" on the second story when they had been called "a girl and a maid" on the first story is one example. Identification of relationships reflects a view of people as less isolated and implies a belongingness not present in a relationship of employer-employee. The expression of a familial relationship where this was not given before was regarded as indicating less autism, egocentricity and withdrawal than when objects and people were merely classified according to occupational role. This latter behavior was looked on as a barrier against closeness to others (16).

D. Change in the direction of less withdrawal from people was noted where individuals in the picture were described as participating in an activity together. Mutual activity and interaction between people in a second story when one figure had been the sole focus of interest previously suggested an increase in responsiveness to others. Consideration of all the characters in a picture and assigning roles to each where previously one character was merely the passive recipient of action from another person suggested a greater awareness of personal roles and worth. Greater potentiality for interaction, for example, is implied in the statement "she is talking to her" than the statement "she is looking at her." The ability to perceive others and report the thoughts and feelings and to deal with a "me—you" relationship of a diad rather than concentrating on either "me" or "you" as a single or isolated individual apart from others was regarded as evidence of less withdrawal from others.

II. Positive changes in attitude toward the world.

A. Dealing with the world on a reality level was taken as evidence of acceptance of the world. During the acute withdrawal phase of the patient's illness the world is rejected because it appears to the patient as hostile and threatening. This should be reflected in the content of the stories, regardless of their theme. Since most of the TAT cards elicit morbid and unhappy stories (5), the tone of the story itself is not expected to change, but less fear and hostility are expected with improvement (6). A change in a story from "the man is being attacked and robbed" to "he is sick" indicates decrease in the hostility perceived as inherent in the environment, and this was rated a positive change.

B. Dealing with the world on a reality level recognizes that a given situation has antecedent and subsequent conditions. Emphasizing only the "here and now" and evading the past and future implications of an activity indicates rejection of the normal flow and continuity of activities. Dealing with past, present, and future is required in the test instructions. Inability or unwillingness to do this by keeping the story in the narrow present further indicates rejection of the world, since the test instructions are a part of the immediate world with which the patient is dealing. With clinical improvement the patient is expected to have a better grasp on reality and will show this by being less restricted to the present. Diminished fear of the world and his own environment is shown by greater compliance with test instructions and by dealing with the normal sequence of time in terms of past, present, and future. Changes in these directions were rated as positive.

C. The ability to give an outcome for a story, whether pleasant or unpleasant, was also evidence of a more realistic view of the world as it exists. Hedging about an outcome or

saying there can be no outcome was considered part of rejection and withdrawal from the world. For example, less fear of the world and more realistic acceptance of it was shown by the conclusion "they'll never see each other again" or "she'll die," as opposed to an earlier statement to the same card "I don't know what the outcome will be."

D. Greater consistency in the logical sequence of the story and integration of relevant details in a picture was looked on as evidence of greater ease and less fear in dealing with the world. Greater coherence and logic in a story indicated that the patient was no longer immobilized by his fear of the environment and could invest his energy in tasks beyond his immediate needs. An example was seen in the conclusion of a story, "the outcome is that he learns from experience," at the second testing instead of the less realistic conclusion given when the patient was acutely ill, "I'm an optimist, so I'll say the outcome will be good" even to a story which was extremely morbid.

III. Positive changes in the expression and control of emotion

A. The apathy and indifference sometimes seen in acutely ill schizophrenic patients is looked on as evidence of withdrawal of emotion rather than its absence. With clinical improvement it is expected that emotions will be expressed more appropriately. A change in the pair of stories to a selected TAT card in the direction of freer and easier expression of emotion was considered a positive change, especially since in the administration of the test the patient was asked to tell what the people were thinking and feeling. If, in the first story, there was no mention of any feeling or emotion, and these were mentioned in the second, the change was called positive and indicative of increased ability to express emotion.

B. Coming to grips with emotion or feeling is manifest not only by giving the emotion or feeling a name, but also by using it effectively and realistically in a given situation. If the patient recognized in one story that emotions motivate behavior, regardless whether the emotion was happy or unhappy, while in an earlier story he merely described behavior, the change was considered positive. The statement "he is leaving home because he is unhappy there" shows more realistic use of emotion than saying "he is leaving home." All changes, in a pair of stories to the same card, in the direction that showed less avoidance of emotion and more realistic and appropriate use of emotion were considered evidence of lessened withdrawal.

Procedure

The population studied consisted of 45 consecutive hospital admissions of cooperative, acutely ill schizophrenic patients between the ages of 16 and 40. Each patient in this study was tested when he was acutely ill and when he left the hospital either as improved or nonimproved. Each individual was both an experimental subject and his own control, since changes between first and second sets of stories were the data analyzed. This population of patients was the same one studied in the analysis of changes on Rorschach indices with clinical improvement (7). The TAT was administered the day following the administration of the Rorschach test.

Because the concept of withdrawal used in this study emphasizes interpersonal relations, the TAT cards used were those in the 1943 series (13) in which more than one person is depicted. Card 18BM was included in the cards selected for this study because the presence of the extra hands suggests the presence of a second person in the scene. The fifteen cards used were numbers 2, 7BM, 8BM, 4, 7GF, 10, 6BM, 9BM, 18GF, 9GF, 6GF, 12F, 18BM, 13MF and 12M. Both male and female subjects were given each card because interpersonal relations involve the patient's attitudes toward both male and female figures. Murray's contention that male and female subjects are more prone to project on the cards designated for each sex has not been substantiated (23).

Two psychologists unfamiliar with the patients rated each pair of stories for a given card according to the general criteria given above. These raters worked independently of each other and of the writer. Each rater had written instructions to guide him in his evaluation of each pair of stories for each patient. The raters knew only that the patients were a group of acutely ill schizophrenic hospital-

ized patients, some of whom had shown improvement before discharge. The raters indicated whether there was positive change or no change in comparing the first and the second stories for each of the 15 TAT cards for each patient.

Treatment of Data

The agreement between two raters in regard to the positive changes or no changes on each card for each patient was analyzed. In order to determine whether the classifications of change or no change were independent, and did not arise by chance (12), chi-square tests were used. The agreements between the raters regarding the changes in the three areas of withdrawal were found to be significant for six of the 15 cards at less than the .05 level These were cards 2, 7BM, 8BM, 10, 6BM and 6GF. The P values for all 15 cards determined from the raters' agreements for each area of withdrawal are given in table 1. Since it can be inferred that the agreement between the raters is not chance agreement, the inference can also be made that the changes in the responses on the stories to the six cards represent changes in the patients' perceptions and attitudes. Of the remaining nine cards, there was agreement that two (7GF and 12M) appear sensitive to changes in perception of people and of emotions. There was agreement that card 4 reflects changes in the perception of the environment and of people, cards 9GF, 9BM and 12 reflect changes in perception of people only, while card 13MF reflects changes in the ability to deal with emotion only. There was no significant agreement on cards 18GF and 18BM in regard to the changes in any area of withdrawal.

TABLE 1.—P Values Derived from Chi-Square Test of the Agreements of Two Raters Regarding Changes in Perception of People, the World, and Emotions

TAT Card	*People*	*Environment*	*Emotions*
2	.05	.05	<.01
7 BM	<.001	<.05	<.05
8 BM	<.001	<.02	<.05
4	<.001	<.05	NS*
7 GF	<.02	NS	<.05
10	<.05	<.02	<.05
6 BM	<.05	<.001	<.001
9 BM	<.05	NS	NS
18 GF	NS	NS	NS
9 GF	<.01	NS	NS
6 GF	.02	.001	.05
12 F	.05	NS	NS
18 BM	NS	NS	NS
13 MF	NS	NS	<.01
12 M	<.02	NS	<.05

* Not significant.

Further statistical tests were made to determine the reliability of the ratings. The six cards which showed general agreement regarding changes in the three withdrawal areas were used in this analysis. The number of changes on the six cards shown by each patient was computed for each area of withdrawal and each patient was ranked according to the number of positive changes he showed. Using the rank order formula $\text{rho} = 1 - \frac{6\Sigma d^2}{N(N^2-1)}$ (12), the relationship found between the ranks given by the two raters for changes in perception of people, in perception of the world, and in the ability to deal with emotion were computed. The obtained correlation values were significantly different from zero, with P values less than .001. The obtained reliability coefficient indicates that the two raters showed better than chance agreement in their evaluation of changes.

The next analysis checked the over-all agreement of the raters on the changes on the six cards for the total sample divided into the three groups of improved, markedly improved, and nonimproved cases. The division of the cases into these three clinical classifications was made on the basis of ratings of the patient's condition at the time of departure from the hospital by two psychiatrists familiar with him during his hospitalization. They agreed that 33 of the 45 cases showed clinical improvement while hospitalized and that 14 of these cases showed marked improvement. The remaining 12 cases showed no clinical change according to both psychiatrists. Those patients who improved and those who did not improve clinically were found to be not significantly different in respect to age, education, length of hospitalization, kinds of therapeutic treatments given and incidence of unhealthy responses on their initial Rorschach protocols (7).

The hypothesis that patients showing remis-

sion from schizophrenic illnesses will not show changes in their TAT stories in the direction of greater interest in and interaction with others, less fear of the world and greater freedom in expressing emotion was tested. Since the best estimate of the true order of the ranks of the subjects was the combined ratings of the two judges, these constituted the data which were analyzed. Using the formula

$$H = \frac{12}{N(N+1)} \Sigma \frac{(R)^2}{n} - 3(N+1),$$

(10), it was found that the rankings of the 14 markedly improved cases and the 12 nonimproved cases were significantly different at the .001 level in each of the three areas of withdrawal. When the ranks of the 19 improved cases were included in the analysis, the differences between the markedly improved, improved, and nonimproved groups were significant at the .001 level with regard to changes in dealing with emotion. The combined ranks on changes in perception of the world as hostile was significant at the .01 level. The ranks of the three clinically defined groups in regard to changes in perception of people was not significant, since P was greater than .10.

From these data it may be inferred that the combined ranks based on the number of changes in the direction of freer expression of emotion and also for less fear of the world discriminate between all improved and nonimproved groups with greater than chance expectations on the six cards together. The combined ranks pointing to increased interest in others or acceptance of others has doubtful value in discriminating between the improved and nonimproved cases. The combined ranks of the two judges clearly distinguish the markedly improved and nonimproved cases in the three areas of withdrawal.

Discussion

The literature on the TAT contains many different schemes for "scoring" and interpretation. In the initial work with the TAT, Murray (13) put forth a scheme for analysis involving the hero, needs, press, thema and outcome. Others have found this scheme too complex for clinical use and have developed other schemes for scoring and/or interpretation. Some are derivative from Murray's type of analysis and some are independent from his. Examples of derivative schemes are found in Stein's work (20). Stein deals with seven areas for analysis: hero, environmental stimuli, hero's behavior, cathexes (objects, activities, people, ideas), inner states, manner of fantasy expression and outcome. Wyatt (24) gives 15 variables which should be studied for successful TAT analysis. Rotter (18) suggests that the stories be read as a whole for a general impression and knowledge about identifications. Then the contentual and structural characteristics of the stories and the familial, social, sexual and general attitudes should be analyzed separately. Rapaport (16) bases his interpretation on the content of the stories, the prevailing tone, figures in the story, strivings and attitudes expressed and obstacles and barriers evident in the content. Terry (21) deals with the plot of the story. In her treatment of the "level of response," which she defines as the degree to which the subject is judged to have become involved in his story, she deals with the feelings apparent in the story.

Other workers have treated TAT stories in either atomistic or holistic ways (4). An example of the atomistic approach is seen in the work of Balken and Masserman (2) in which they count the words representing different parts of speech and compare their incidences in different diagnostic groups. Bellak (3) objects to this approach and points out that an holistic approach is necessary to understand the TAT stories of patients. Murray (14) also objects to emphasis on formal characteristics of the stories. Murray further states that the stories are not related to overt behavior. In contrast to this view, Arnold (1) looks on each TAT card as a sample situation to which the subject reacts as he would in a real situation, and considers the responses to the situations rather than the individual traits inferred from the stories as the important TAT data. She takes the holistic view when she emphasizes the integrated content of the stories as the crucial material obtained from a TAT analysis. The test, according to Arnold, deals with present dominant conflicts and the people involved in them. The people in the story are also emphasized in the type of analysis expounded by Terry (21).

In reviewing the growing literature on the

TAT, Holt (9) and Watson (23) point out that there is little agreement in regard to methods of analysis and interpretation but that all workers make use of a general inspection of the stories. It can be said that some schemes emphasize the structure of a story and its formal features, while other schemes emphasize the content only. However, there appears to be general agreement among clinicians that

"a sentence-by-sentence, word-by-word dissection of a subject's productions giving equivalent importance to every utterance without regard to the properties of the fantasy-evoking stimuli and/or their valence for a large number of individuals is not a valid procedure" (17, p. 156).

The approach in this study is essentially holistic and in keeping with clinical use of the TAT. Reactions to people, the environment and the emotional life of the story teller are the general areas which are examined by the TAT worker, though these areas of behavior have been classified in different ways by different writers. The generalized concept of perception of the world as it is used in this study is related to the categories of press (13) and environmental stimuli (20). The attitude toward people is used in more sharply defined and specific ways by Arnold (1), Terry (21), Rotter (18) and others. The general area of ability to handle emotions is similar to the emphasis on inner states by Stein (20), the prevailing tone of the story by Rapaport (16) and outcome by Murray (13).

The three generally described areas in which withdrawal diminishes with clinical improvement were successfully identified on six TAT cards by two raters working independently. These cards are more matter-of-fact and clearly drawn pictures than the other cards used. They also seem less directly focussed on specific traumatic areas or toward specific problems. It would appear that the somewhat bizarre portraiture in 12F of the "weird old woman," in 12 M of the "gaunt form of an elderly man," or the suggestive figure of the nude female figure in bed in 13MF or the violence in 18GF and the surprising extra hands in 18BM may work against identification and projection by the patient since these scenes and characters are foreign to everyday experience. The common qualities in the pictures where the agreement by the raters was significant suggests that for diagnostic and interpretive purposes it may be well to use pictured situations where the stimulus is not too dramatic and is likely to be within the realm of the patient's own experience.

The agreement between the judges regarding positive and no changes suggests that comparisons of pairs of stories to the same card at different times during the course of an illness is not only feasible but of value in understanding change in a patient's condition. The findings also suggest that repeat reliability studies are possible with this test. Significant changes in TAT stories appear to be present when there are changes in the patient's general condition. This is consistent with the findings by Tomkins (22) who found changes in stories after short periods of time when the personality was changing rapidly, while no changes were found in one case with a very rigid personality after many repeated TAT administrations.

The changes in attitudes were reflected by the TAT stories which were treated holistically. These changes in attitudes were recognized without emphasizing the themes or content or the diagnostic and dynamic implications of the stories. These findings suggest that it is feasible in research to use TAT stories globally within an explicit conceptual framework.

Summary and Conclusions

Three areas in which withdrawal changes when there is remission from an acute schizophrenic illness can be identified in stories given to six selected TAT cards. The three areas concern attitudes toward people (interpersonal relations), perception of the world and the ability to handle emotions. The combined ratings of changes on repeated TAT stories by two independent raters clearly differentiate cases showing marked improvement from nonimproved ones. Greater acceptance of people, less fear of the world and better ability to accept emotions are shown on the second sets of TAT stories by the markedly improved patients. In the cases with undifferentiated improvement the judgments of change discriminate between the improved and nonimproved groups in regard to perception of the world and ability to handle emotions but are not effective in indicating change in perception of others (interpersonal relations).

The results strongly suggest that the less bizarre and more realistic pictures elicit stories which reflect a patient's changed perception more successfully. They also suggest that it is advisable to give the same set of TAT cards to a given patient more than once during his hospitalization to evaluate his clinical progress. The findings in this study indicate that perception of the pictures changes when there is change in the patient's clinical condition.

This study suggests a method by which carefully selected cases may be used to increase knowledge about the reliability and the validity of the TAT. A method for analyzing the TAT productions using empirically validated clinical concepts, such as withdrawal, instead of emphasizing atomistic or formal story characteristics, is meaningful both for practical work and for clinical research.

REFERENCES

(1) Arnold, M. B.: A demonstration of analysis of the TAT in a clinical setting. *J. Abnorm. & Social Psychol., 44:* 97-111, 1949.

(2) Balken, E. R., and Masserman, J. H.: The language of phantasy, III. The language of the phantasies of patients with conversion hysteria, anxiety state, and obsessive-compulsive neuroses. *J. Psychol., 10:* 75-86, 1940.

(3) Bellak, L.: Thematic Apperception Test and other apperceptive methods. In *Progress in Clinical Psychology,* Brower, D. and Abt, L. E., Eds., Vol. I, Ch. 9, 149-172. New York: Grune and Stratton, 1952.

(4) Davenport, B. F.: The semantic validity of TAT interpretations. *J. Consult. Psychol., 16:* 171-175, 1952.

(5) Eron, L. D.: Frequencies of themes and identifications in the stories of schizophrenic patients and non-hospitalized college students. *J. Consult. Psychol., 12:* 387-395, 1948.

(6) Garfield, S. L., and Eron, L. D.: Interpreting mood and activity in TAT stories. *J. Abnorm. & Social Psychol., 43:* 338-345, 1948.

(7) Goldman, R.: Changes in Rorschach performance and clinical improvement in schizophrenia. Unpublished Ph.D. dissertation, Boston University, 1955.

(8) Henry, W. E.: The Thematic Apperception Technique in the study of group and cultural problems. In *An Introduction to Projective Techniques,* Anderson, H. H. and Anderson, G. L., Eds., Chapter 8. New York: Prentice Hall, 1951.

(9) Holt, R. R.: The Thematic Apperception Test. In *An Introduction to Projective Techniques,* Anderson, H. H., and Anderson, G. L., Eds., Chapter 7. New York: Prentice Hall, 1951.

(10) Kruskal, W. H., and Wallis, W. A.: Use of ranks in one-criterion variance analysis. *J. Am. Statistical A., 47:* 583-621, 1952.

(11) Mayman, M., and Kutner, B. Reliability in analyzing Thematic Apperception Test stories. *J. Abnorm. & Social Psychol., 42:* 365-368, 1947.

(12) McNemar, Q.: *Psychological Statistics.* New York: John Wiley & Sons, Inc., 1949.

(13) Murray, H. A.: *Thematic Apperception Test.* Cambridge, Mass.: Harvard University Press, 1943.

(14) Murray, H. A.: Uses of the thematic apperception test. *Am. J. Psychiat., 107:* 577-581, 1951.

(15) Murray, H. A.: Thematic Apperception Test. In *Contributions Toward Medical Phychology,* Weider, A., Vol. II, 636-649. New York: The Ronald Press Company, 1953.

(16) Rapaport, D.: *Diagnostic Psychological Testing,* Vol. II. Chicago: Year Book Publishers, 1946.

(17) Ritter, A. M., and Eron, L. D.: The use of the Thematic Apperception Test to differentiate normal from abnormal groups. *J. Abnorm. & Social Psychol., 47:* 145-158, 1952.

(18) Rotter, J. B.: Thematic Apperception Tests: suggestions for administration and interpretation. *J. Person., 15:* 70-92, 1946.

(19) Schneidman, E. S.: The TAT Newsletter. *J. Proj. Tech., 18:* 111-119.

(20) Stein, M. I.: *The Thematic Apperception Test: An introductory manual for its clinical use with male adults.* Cambridge, Mass.: Addison-Wesley Press, Inc., 1948.

(21) Terry, D.: The use of a rating scale of level of response in TAT stories. *J. Abnorm. & Social Psychol., 47:* 507-511, 1952.

(22) Tomkins, S. S.: *The Thematic Apperception Test: The theory and technique of interpretation.* New York: Grune and Stratton, 1947.

(23) Watson, R.: *The Clinical Method in Psychology.* New York: Harper Brothers, 1951.

(24) Wyatt, F.: The scoring and analysis of the Thematic Apperception Test. *J. Psychol., 24:* 319-330, 1947.

TRANSFERENCE AND THE PHYSICAL PRESENCE OF THE PHYSICIAN

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An understanding of the mechanisms of psychotherapy involves a clarification of the role of the transference relationship. Transference is first mentioned in the mongraph on hysteria by Freud and Breuer (6). The first discussion of transference per se was in the case of Dora where Fread wrote, "Transferences . . . are new editions or facsimiles of the tendencies and phantasies which are aroused and made conscious during the progress of the analysis; but they have this peculiarity, which is characteristic for their species, that they replace some earlier person by the person of the physician" (23, p. 139). Freud also noted that the transference may take advantage of "some real peculiarity of the physician's person." In his Introductory Lectures, Freud wrote of the transference neurosis as a new illness which supplements the earlier set of neurotic symptoms: ". . . the conquest of this new, artifically acquired neurosis coincides with the removal of the illness which existed prior to the treatment, that is, with the accomplishing of the therapeutic task" (20, p. 386). Other works by Freud amplify these definitions of transference and transference neurosis (21, 22, 24, 25).

Freud's concept of transference and transference neurosis has undergone modifications on clinical and theoretical grounds. An understanding of the way these concepts are used currently requires an appreciation of the nuances of modification rather than a series of radical alterations. These alterations occur in the literature on the following topics: character analysis (45), repetition compulsion (8, 10, 34, 38, 40, 43), hypnosis and suggestion (11, 35, 44), "active therapy" (12, 35), corrective emotional experience (2, 3), analytic ego psychology (1, 54, 55), defense transference (19, 27), technique of analysis (29, 30), "acting" in the transference (19, 31, 47), pre-oedipal object relations (37, 50, 51), interpersonal school of psycho-analysis (17, 46, 56), transference in children (18, 36), transference in schizophrenics (26), and countertransference (4, 7, 28, 33, 58). Interesting variations in the concept of transference are expressed by Silverberg (52), Meerloo and Coleman (42), and Maccalpine (41). Clinical reports by various workers (5, 32, 48, 49) have served to sharpen some of the theoretical formulations about transference and transference neurosis. Several detailed historical accounts of the concept of transferences have appeared (9, 39, 41). The statistical study of the transference relationship has received relatively little attention; however, one worker (13–16) has used the Q-technique design in his studies of transference.

The elements leading to the development of transference and transference neurosis are still a source of controversy. This study is an exploratory approach to one aspect of this problem by the removal of one variable from the psychotherapeutic situation: the physical presence of the physician.

This study must provide a definition of transference. It consists of conscious and unconscious, repetitious, emotional reactions of varying complexity on the part of the patient toward the physician in the therapeutic situation. These emotional reactions represent needs often disguised in phantasy and are due to all the earlier and current emotional respone patterns of the patient, especially his earliest object relations, and are also due to realities of the therapeutic situation. Transference is most

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Acknowledgement is made to David A. Wasser, M.D., Max C. Pepernik, M.D., and Robert M. Powell, M.D., who treated patients in this project.

This material is taken in part from a thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in the Department of Psychiatry and the Graduate College of the State University of Iowa (Dr. Lowinger). This author is now at the U. S. Public Health Service Hospital, New Orleans, Louisiana.

readily demonstrated when the real figure of the physician is replaced by one or more earlier significant figures in the patient's life.

The physical arrangement used in this study consisted of two adjacent, sound-proof rooms. The room for the patient had in one wall a rectangular mirror which was a one-way observation glass. The patient's room also contained a loud speaker mounted on the wall and a microphone. The physician's room contained the electrical equipment, consisting of two vacuum tube amplifiers for two-way communication. There was a microphone which communicated with the loud speaker in the patient's room and a set of earphones connected to the microphone in the patient's room. The physician's room was arranged so that he could listen and talk to the patient while observing him through the one-way mirror. The electrical equipment furnished satisfactory quality reproduction of both the voice of the patient and the physician.

The patients selected for the study came from the out-patient service with the exception of one hospitalized patient. They represented neurotic problems which were considered amenable to psychotherapy after a diagnostic workup. They physical arrangement of the experimental situation was explained to each patient by the interviewing physician before he was asked if he would be willing to receive psychotherapy in this setting. All the patients used gave consent to be treated in this fashion. Each patient was told that psychotherapy would follow conventional lines with the exception that he would not be in the same room as the physician or see him at any time. Each patient was also told that the patient-physician relationship was of particular interest in his treatment. It was further explained that the physician would be known to the patient by a pseudonym. No comment was made to the patient as to whether the physician would see or observe the patient and no patient raised this question. A nurse conducted the patient from the waiting room to the treatment room at the time of appointment.

The four therapists were male resident physicians. The psychotherapy was directed toward the goal of relieving the important presenting symptoms. This objective was established only after evaluation of the descriptive and dynamic material allowed a tentative formulation of such a goal. Different psychotherapeutic techniques were used, including free association, directed association, dream and phantasy interpretation, and "working through" resistances. These techniques varied with the patients and with the physicians. Every effort was made to understand the transference, which was not interpreted to any great extent in most instances because it did not constitute an obstacle in psychotherapy.

The methods for the evaluation of certain aspects of the transference gradually emerged during the project. Methodology remained a major concern and thus flexibility in this area was appropriate to this pilot study. Each patient's production were evaluated in terms of the transference. This included all verbal as well as non-verbal behavior in the therapy room. This was supplemented by questions on the transference relationship at least as frequently as every third interview and more often if appropriate. The subjectivity of these measures called for some objective counterpart. Sample questions were: What is your impression of the personality of your doctor? Of his appearance? How do you feel about him? Does he remind you of anyone you have known previously? In what way? The first three patients each commented in writing on questions about their transference attitude following each session. However, the written comments tended to add little to the verbal report of the patients, so this method of evaluation was discontinued. An attempt at objectifying the evaluation of the transference in the last seven cases was made by means of an evaluation interview with the patient by a senior staff physician.

Five men and five women were treated; they ranged in age from 25 to 38 years. Treatment was one to three times a week, averaging a total of 31 hours over an average of 29 weeks. Follow-up on six patients who have been out on psychotherapy for more than three months averaged 12 months.

The results demonstrate that psychotherapy can be done without the physical presence of the physician. Of ten patients two had a remission of their main complaints, four were improved, and four were unchanged in terms of their entrance complaints.

It has seemed, within our framework of reference, that the physical presence of the therapist is not necessary for the development of a transference relationship. Eight of the ten patients formed a transference relationship within the terms of the definition of transference. This serves to emphasize that the verbal productions of the physician and the needs of the patient in the therapeutic setting are sufficient to form a transference.

There is a difference, however, between the transference relationship in the experimental situation and the transference in the conventional psychotherapeutic situation where the patient and physician are in the same room. The qualities of this difference are not readily discerned from the methods and results of this study. A quantitative distinction does emerge. There appears to be a reduction in the intensity of the various affects that compose the transference with the physical absence of the therapist. This dilution of the transference relationship means something quite different in each patient in terms of the over-all influence on resistances, psychotherapeutic course, and the results as a whole. This reduction in the intensity of feelings composing the transference relationship is shown by the failure of any of the ten patients treated in the experimental situation to utilize the transference relationship as a specific resistance to psychotherapy.

A 25-year-old male graduate student, whose entrance complaints were anxiety and the inability to ejaculate during masturbation, was treated for 45 hours over an eight-month period without evidence of transference reactions which impeded psychotherapy. This patient's profound characterological difficulties included passive-aggressive conflicts, unconscious and unacceptable homosexual strivings, and a feminine identification, all of which appear to be related to an oedipal situation. The physician who was treating this patient was simultaneously treating another male university student using the same psychotherapeutic technique for about 50 hours over an eight-month period. The important distinction was that in this latter instance physician and patient were in the same room. This treatment in the conventional therapeutic situation of a comparable patient with similar complaints and remarkably similar nuclear conflict led to definite transference reactions which impeded psychotherapy.

Other physicians treating patients in this study noted the absence of intensified transference reactions in patients in the experimental situation where they felt that they might have been present if the therapist were in the same room as the patient. This is demonstrated in another way by a patient who was treated in the experimental situation for one hour a week over 22 weeks, without an intense transference reaction, but who developed one when treated by the same physician in a conventional psychotherapeutic situation for one hour per week over 14 weeks.

She was a 31-year-old married woman who complained of many symptoms since 1945, including sore throat, fatigue, homicidal impulses toward the husband, tearfulness, depression, suicidal thoughts, "upset stomach," nausea, vomiting, headaches, right-sided pain, pain in the "tailbone" and lower abdominal pain. These symptoms severely limited her social activity and efficiency in housework and also prevented her from working, attending school or having children.

The total picture presented by the patient and her symptomatology was one of a pervasive character disorder with fixation at an infantile level of emotional functioning. There was no definite change in the patient in terms of her presenting symptoms. She acquired some insight into her hostility which she was better able to express. She continued to show little insight into the psychogenic nature of her physical complaints and at times stated that she believed she had a somatic illness that constituted the explanation for these symptoms, along with a mental illness by which she explained her depressive, suicidal, and hostile feelings.

Important psychodynamic factors elicited during therapy included guilt over unacceptable hostility and aggression, illness as a means of obtaining love, the use of somatic symptoms as a defense against sexuality, and feelings of inadequacy and inferiority.

The evaluation of the transference relationship during the 22 hours of psychotherapy in the experimental situation was as follows: Interviews have been characterized by repeated, frequent somatic complaints, although she has

been quiet, calm, and without overt anxiety. The conscious feelings involved in the transference include hostile and negative emotions and also positive feelings. This was in keeping with the patient's ambivalent response to all other persons in her life. The hostile aspects of the transference were more prominent in terms of the material produced. They emphasized fear and dislike of all men and especially authority figures and physicians. This often led to projection of the patient's hostile feelings into the therapist's voice. Conscious positive attitudes were present in the form of compliments and requests for reassurance from the physician. Unconscious ambivalent attitudes in the transference were related to the need for love which had been thwarted in the relationship with the mother and stepfather. No specific unconscious identification of the physician was evident, although the ambivalence, hostility and projection in the relationship with him were typical of all other relationships with both men and women.

The 14 hours of psychotherapy with the patient and the physician together in one room can be summarized in terms of the transference as follows: The patient was quite glad to enter the room with the physician. She stated she no longer was afraid that others were listening and she now felt more secure. The relationship between the patient and the physician immediately became warm, friendly, and personal in character due to a completely different attitude on the part of the patient. She made many comments and asked questions about the physician's activities, personal mannerisms and life outside the hospital with considerably more affect than on any previous occasion. The last eight of these interviews found the patient behaving seductively toward the physician and engaged in erotically-toned "kidding." In this way she was able to suggest humorously that they meet in another city for an affair, to interpret the physician's profession of interest in her somatic complaints as interest in "my body," while at the same time she expressed fears of her obvious sexual impulses toward him. She indicated in the last five interviews that she felt well only during the therapeutic hour and did not mention spontaneously the many mental and physical complaints present throughout the week, although they were still present in the same severity. There was evidence of latent hostility in her erotic behavior toward the physician, much as there had been evidence of latent sexual feelings in her earlier transference relationship which had emphasized overt hostility to the therapist. It was felt by the therapist as well as the evaluating psychiatrist that she had changed markedly in her behavior and was now manifesting distinct evidence of an intensified transference reaction.

These differences in quantity and quality of affective responses by the patient in the absence of and in the presence of the therapist suggest that the relationship of the affective components of psychotherapy might be explored more systematically by controlled procedures where therapy is initiated with or without the therapist present and then this situation reversed. This is suggested particularly since the experimental procedure is weighted in the direction of intellectalization of the treatment and since the conventional therapeutic setting seems to involve more affect.

It was the impression of the physicians who participated in this study that the experimental situation offered a means of learning something unique about psychotherapy. One of the ways in which this was accomplished came about in terms of giving the physician insight into his own reactions to the patient. Since the therapist was alone and in a separate room, it was more acceptable to him to feel and express anxiety, fear, sexual feelings and hostility in relation to the patient. Since there was no reason for the physician to attempt to resemble the conventional stereotype of the attentive psychiatrist, he was free to become increasingly aware of and to express various emotions such as boredom or exasperation by his motor behavior in the room. One physician reported an increased awareness of his own fears and hostility in response to a patient who verbalized his general allegiance to authoritarian, militaristic, and anti-Semitic attitudes. These clues to the therapist's own reactions could then be used to observe the counter-transference.

Unique motor behavior by two of the patients occurred during therapy session which probably would not have been present had the physician and patient been in the same room.

A male patient with latent homosexual conflicts had erections on several occasions in response to sexually stimulating free associations. A 26-year-old married female patient engaged in a combination of seductive and masturbatory behavior on one occasion. She stopped this behavior abruptly when the physician pretended to be called away from his microphone for a short period of time. When the physician announced his return, she resumed this activity. These patients seemed to be unaware of being observed through the mirror by the physician. If they were not completely unaware of being observed, then the total experimental situation offered a unique kind of permissiveness for the discharge of motor behavior. The other eight patients manifested no unusual motor behavior during the interviews in the experimental situation.

The relationship of such overt behavior to the transference in this setting was not determined, nor can its role in the therapy be evaluated. The fact that it did occur, however, in three cases raises interesting questions. Would the impulses back of the behavior have been conscious as readily if the physician had been present in the room with the patient? If the impulses affected the behavior of the patient in minimal degrees through minor changes in bodily attitude, vocal inflection, etc., would this have been noted, perhaps unconsciously, by the therapist and in subtle ways changed the course of the therapeutic process? Would more "acting out" behavior have occurred if the physician had encouraged it and how would this affect therapy? It is likely that persons experienced with psychotherapy would be able to provide a variety of answers to such questions. Our point here is that we believe the experimental technique employed in this study makes possible more objective formulations than when the physician is immersed in the interpersonal psychotherapeutic relationship alone with his patient.

The verbalizations of the patient and the physician in the therapy situation represent the more cognitive aspects of psychotherapy. From this study the physical separation of physician and patient serves to remove an important source of some of the emotional stimuli of the therapeutic situation. This serves to emphasize the view that the transference relationship is based on the patient's needs in terms of his early and current behavior patterns rather than being caused by stimuli originating from the therapist.

Some observations on the experimental technique by the patients are of interest. In general, most of the patients did not object to the procedure and accepted it quite readily. Some expressed a feeling of strangeness initially about talking to a person they could not see. One patient commented that she believed the technique helped her concentrate on her problems since she would not have to worry about getting involved with another person. The patient, discussed earlier, who was treated both in the experimental setting and in the conventional manner, objected frequently to the absence of the physician. She said she couldn't really talk about herself with a voice. It was this patient who behaved seductively toward the physician when brought into his presence. These observations highlight the important differences from patient to patient, related to his problems and his personality structure and dynamics which may promote or retard the development of transference, counter-transference, transference neurosis, and the course and outcome of treatment. They suggest that the experimental technique might be the preferable method with some patients, while on the other hand, the conventional method may be required in some patients.

Summary

This project is an exploratory study of transference in psychotherapy by the removal of one variable: the physical presence of the physician. The results are based on ten patients with neurotic problems treated with psychotherapy in the experimental situation. The conclusions are as follows.

1. Psychotheraphy can be done with only verbal communication when the patient and physician are in separate rooms. Of ten patients treated by this experimental technique, two had a remission of symptoms, four were improved, and four were unchanged.

2. A transference relationship develops under the experimental conditions. This demonstrates that the transference relationship is not dependent on the non-verbal aspects of the behavior and attributes of the psychotherapist.

3. The transference relationships which develops under the experimental conditions is less emotionally or affectively intense than the transeference relationship would be with the patient and the physician in the same room. The more intense reactions developed with the physician present may form barriers to the progress of treatment of certain patients by brief dynamic psychotherapy.

4. The experimental technique allows the physician a better understanding of the countertransference by increasing his awareness of his own behavior in the therapeutic situation.

5. The methods employed furnished a profitable approach to the attempt to objectify the transference relationship. These methods can be modified and refined so as to permit further study of the nature and mechanism of transference.

BIBLIOGRAPHY

(1) Alexander, F.: The problem of psychoanalytic technique. *Psychoanalytic Quart., 4:* 588-611, 1935.

(2) Alexander, F.: *Fundamentals of Psychoanalysis,* pp. 312. New York: W. W. Norton. and Co., Inc., 1948.

(3) Alexander, F., and French, T.: *Psychoanalytic Theraphy,* xiii and 353 pp. New York: Ronald Press Company, 1946.

(4) Balint, A., and Balint, M.: On transference and counter-transference. *Int. J. Psycho-Anal., 20:* 223-230, 1939.

(5) Bibring-Lehner, G.: A contribution to the subject of transference resistance. *Int. J. Psycho-Anal., 17:* 181-189, 1936.

(6) Breuer, J., and Freud, S.: *Studies in Hysteria,* ix and 241 pp. New York: Nervous and Mental Disease Publishing Company, 1936.

(7) Cohen, M. B.: Countertransference and anxiety. *Psychiatry, 15:* 231-243, 1952.

(8) Fenichel, O.: Symposium on the theory of therapeutic results of psycho-analysis. *Int. J. Psycho-Anal., 18:* 133-138, 1937.

(9) Fenichel, O.: Problems of psychoanalytic technique. *Psychoanalyt. Quart., 8:* 438-470, 1939.

(10) Fenichel, O.: *The Psychoanalytic Theory of Neurosis,* x and 703 pp. New York: Norton and Company, 1945.

(11) Ferenczi, S.: *Contributions to Psycho-analysis,* iv and 288 pp. Boston: Richard G. Badger, 1916.

(12) Ferenczi, S.: *Further contributions to the theory and technique of psycho-analysis,* 473 pp. London: Hogarth Press and Institute of Psycho-Analysis, 1926.

(13) Fiedler, F.: The concept of the ideal therapeutic relationship. *J. Consult. Psychol., 14:* 239-245, 1950.

(14) Fiedler, F.: A comparison of psycho-analytic nondirective, and Adlerian therapeutic relationships. *J. Consult. Psychol., 14:* 436-445, 1950.

(15) Fiedler, F.: A method of objective quantifications of certain countertransference attitudes. *J. Clin. Psychol., 7:* 101-107, 1951.

(16) Fiedler, F., and Senior, K.: An exploratory study of unconscious feeling reactions in fifteen patient-therapist pairs. *J. Abnorm. & Social Psychol., 47:* 446-453, 1952.

(17) Fleming, J.: Observations on the defense against a transference neurosis. *Psychiatry, 9:* 365-374, 1946.

(18) Freud, A.: *The Psycho-analytical Treatment of Children,* xii and 98 pp. London: Imago Publishing Company, 1946.

(19) Freud, A.: *The Ego and the Mechanisms of Defense,* x and 196 pp. New York: International Universities Press, 1946.

(20) Freud, S.: *A General Introduction to Psychoanalysis,* 412 pp. New York: Perma Giants, 1920.

(21) Freud, S.: On the history of the psycho-analytic movement. *Collected Papers,* Vol. I, 287-359. London: Hogarth Press and Institute of Psychoanalysis, 1924.

(22) Freud, S.: Further recommendations in the technique of psycho-analysis. *Collected Papers,* Vol. II, 342-365. London: Hogarth Press and Institute of Psycho-Analysis, 1924.

(23) Freud, S.: Fragment of an analysis of a case of hysteria. *Collected Papers,* Vol. III, 13-146. London: Hogarth Press and Institute of Psycho-Analysis, 1924.

(24) Freud, S.: *Beyond the Pleasure Principle,* 90 pp. London: The Hogarth Press and the Institute of Psycho-Analysis, 1922.

(25) Freud, S.: *New Introductory Lectures on Psycho-Analysis,* xi and 257 pp. New York: W. W. Norton and Co., Inc., 1933.

(26) Fromm-Reichmann, F.: Transference problems in schizophrenics. *Psychoanalyt. Quart., 8:* 412-425, 1939.

(27) Gitelson, M.: Intellectuality in the defense transference. *Psychiatry, 7:* 73-86, 1944.

(28) Gitelson, M.: The emotional position of the analyst in psycho-analytic situation. *Int. J. Psycho-Anal., 33:* 1-10, 1952.

(29) Glover, E.: Lectures on technique in psychoanalysis, Part 1 and 2, *Int. J. Psycho-Anal., 8:* 311-338, 1927.

(30) Glover, E.: Lectures on technique in psychoanalysis, Part 3 and 4. *Int. J. Psycho-Anal., 8:* 486-520, 1927.

(31) Greenacre, P.: General problems of acting out. *Psychoanalyt. Quart., 14:* 455-467, 1950.

(32) Hannett, F.: Transference reactions to an event in the life of the analyst. *Psychoanalyt. Rev., 36:* 69-81, 1949.

(33) Heiman, P.: On counter-transference. *Int. J. Psycho-Anal., 31:* 81-84, 1950.

(34) Horney, K.: *New Ways in Psychoanalysis,* 313 pp. New York: W. W. Norton and Co., Inc., 1939.

(35) Jones, E.: *Papers on Psycho-Analysis,* 4th Ed., x and 643 pp. New York: William Wood and Co., 1938.

(36) Klein, M.: *Contribution to psycho-analysis,* 416 pp. London: Hogarth Press, Ltd., and the Institute of Psycho-Analysis, 1948.

(37) Klein, M.: The origins of transference. *Int. J. Psycho-Anal., 33:* 433-438, 1952.

(38) Kubie, L.: A critical analysis of the concept of a repetition compulsion. *Int. J. Psycho-Anal., 20:* 390-402, 1939.

(39) Lagache, D.: Le problème du transfert. *Rev. franc. Psychanal.,* 14th Conférence des Psychanalysts de Langue française, Paris, 1 novembre, 1951.

(40) Lagache, D.: Some aspects of transference. *Int. J. Psycho-Anal., 24:* 1-10, 1953.

(41) Macalpine, I.: The development of the transference. *Psychoanalyt., Quart., 19:* 501-539, 1950.

(42) Meerloo, J., and Coleman, M. L.: The transference junction: a study of normal and pathological transference. *Psychoanalyt. Rev., 38:* 205-221, 1951.

(43) Nunberg, H.: Symposium on the theory of therapeutic results of psycho-analysis. *Int. J. Psycho-Anal., 18:* 161-189, 1937.

(44) Oberndorf, C.: Constant elements in psychotherapy. *Psychoanalyt. Quart., 15:* 435-449, 1946.

(45) Reich, W.: *Character Analysis,* xxii and 328 pp. New York: Orgone Institute Press, 1945.

(46) Reich, J.: The transference phenomenon in psychoanalytic therapy. *Psychiatry, 6:* 147-156, 1943.

(47) Roth, R.: The acting out of transference. *Psychoanalyt. Rev., 39:* 69-78, 1952.

(48) Rothenberg, S.: Transference situations in an hour of analysis. *Psychoanalyt. Rev., 32:* 340-352, 1945.

(49) Saul, L.: A note on the telephone as a technical aid. *Psychoanalyt. Quart., 20:* 287-290, 1951.

(50) Schmideberg, M.: The mode of operation of psycho-analytic therapy. *Int. J. Psycho-Anal., 19:* 310-320, 1938.

(51) Sharpe, E. F.: The technique of psycho-analysis. *Int. J. Psycho-Anal., 11:* 251-277, 1930.

(52) Silverberg, W.: The concept of transference. *Psychoanalyt. Quart., 17:* 303-321, 1948.

(53) Stern, A.: On the counter-transference in psychoanalysis. *Psychoanal. Rev., 11:* 66-74, 1924.

(54) Strachey, J.: The nature of the therapeutic action of psychoanalysis. *Int. J. Psycho-Anal., 15:* 127-159, 1934.

(55) Strachey, J.: Symposium of the theory of the therapeutic results of psycho-analysis. *Int. J. Psycho-Anal., 18:* 139-145, 1937.

(56) Thompson, C.: Development of awareness of transference in a markedly detached personality. *Int. J. Psycho-Anal., 19:* 299-309, 1938.

(57) Thompson, C.: Transference as a therapeutic instrument. *Psychiatry, 8:* 273-378, 1945.

(58) Winnicott, D.: Hate in the counter-transference. *Int. J. Psycho-Anal., 30:* 69-74, 1949.

STUDIES IN ALPHA BLOCKING FOLLOWING ELECTRO-CONVULSIVE THERAPY[1]

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Investigators have made extensive studies of the changes in the electroencephalogram (1–3, 10, 13–15) and the changes in the psychological processes (6, 7, 11) following administration of electrically induced convulsions. They have been unable to make significant correlations between these two sets of studies (8, 9, 12).

The purpose of our experiment was to determine the effects of problem solving on the dominant activity of the cerebral cortex as recorded by the electroencephalogram both before and following electrically induced convulsions. We hoped that this study would enable us to make significant correlations between physiological activities, as represented by the electroencephalogram, and psychological processes.

Characteristics of Alpha Activity

Effect of psychological processes

In man, thinking, when considered in the broadest sense, either includes or is associated with the processes of attention, perception, retention, recall, analysis and synthesis. Thinking processes occur in the so-called normal man and are included as part of the descriptions of psychopathological syndromes. In other words the thinking process and the psychopathological syndrome are two separate frames of reference.

Alpha activity can be related to the thinking processes in two ways. First, alpha can be related to what we have called non-goal-directed thinking. This is characterized thus: a) the individual is in the resting state, b) if asked, he can report a variety of thoughts, and c) the alpha activity assumes its typical pattern. Second, alpha can be related to what we call goal-directed thinking, i.e. the amount of alpha is reduced in frequency, amplitude, waxing and waning when the individual is performing an arithmetic problem.

The severity of the psychopathology at times seems to determine the degree to which the individual is able to participate in goal-directed activity but otherwise has no direct effect on alpha activity.

Various observers have measured the effects of emotions on alpha activity (4). Although some changes have been noted we feel that their significance is outweighed in importance by the type of thinking involved.

Effects of electrically induced convulsions on alpha activity and psychopathology.

Electrically induced convulsions may cause a shift in frequencies from alpha to lower frequencies, may increase the amplitude and may result in the reduction of the total percentage of alpha. These effects appear to be augmented by the presence of abnormal patterns before electrically induced convulsions were initiated and by increased frequency and the number of electrically induced convulsions.

Electrically induced convulsions affect behavior reactions. Here too, the thinking process and the psychopathology should be considered in two different frames of reference.

Experimental Procedure

Goal-directed thinking produces a change in alpha activity. Electrically induced convulsions produce a change in alpha activity. So, in our experiment the critical data will be the effects of goal-directed thinking on the alpha activity of the individual who has undergone an electrically induced convulsion.

We chose the solving of an arithmetic problem as the goal-directed task because it produced more consistent, significant and reliable changes both before and after induction of electrically induced convulsions, because it

[1] Presented at the meeting of the American Psychiatric Association, May 8, 1953, Los Angeles.

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seemed least affected by psychopathology and because it was easy to administer.

The procedure consisted essentially of performing arithmetic problems before and after electrically induced convulsions while an EEG was taken from a typical six-lead placement of solder electrodes with bentonite paste. A six-channel Grass and an eight-channel Medcraft standard EEG machines were used for the test.

There were 35 patients who received electrically induced convulsions and 17 control subjects in this experiment. Regarding selection of patients, we found that a reasonable amount of clear-cut alpha activity was necessary as well as the ability of the patient to cooperate in the performance of the problem solving.

Twenty-six out of 35 patients were tested 1½ hours after the 2nd, 4th, 6th or 8th and every consecutive 4th electrically induced convulsion. The remaining 9 patients were tested from 11 minutes to 4 hours after the electrically induced convulsions. However, for each patient the time after shock remained constant. Whenever possible, each patient was followed 3 and 6 weeks after electroconvulsive therapy was completed.

The type of problems asked were classified into two groups—simple and complex. There were always 3 simple problems which remained the same, that is 1 plus 1, 2 plus 2 and 3 plus 3. There were 4 complex problems and these consisted of two sets which were given alternately in order to cut down on the learning factor. Set #1 consisted of problems 26 plus 13, 83 plus 19, 14 times 9 and 18 times 12. Set #2 consisted of 23 plus 14, 79 plus 15, 16 times 7 and 17 times 12.

Each subject was told to keep his eyes closed during the entire procedure, that he was going to be asked a group of arithmetic problems, that he should answer out loud, that he could take as long as he wanted to figure out the problems and that a correct answer was not of prime importance as long as he made an effort to figure it out.

The method of scoring went from zero, which meant no reduction at all in the voltage of alpha activity during the problem, to 4, which meant that during the problem solving the voltage fluctuation was reduced to practically a straight line. Intermediate scores, including halves, were used. Each score was checked by at least one other observer.

Analysis of Data

The following observations were made from the observable data. In the initial recordings all the control subjects without psychopathology showed a rather consistent reduction of voltage to a moderate degree with the solving of the simple arithmetic problems. The same groups showed a more pronounced reduction of the voltage when they solved the more complex problems. Since the more complex problems took a longer period of time, the duration of the reduction of the voltage was greater than the simple. Observations concerning the latency before the voltage was reduced and the return to alpha activity during lengthy problem solving are not subjects for discussion now. With problem solving we measured as significant the earliest and most pronounced change regardless of its channel location.

In those initial tracings of the untreated and treated groups with psychopathology the findings were similar to those in the control group except the reduction in voltage both for simple and complex problems was not so consistent. In 4 cases there was practically no reduction of the alpha activity during the solving of the simple problems. But alpha was always reduced with the complex problems.

In the treated group, where the average number of 10 electrically induced convulsions were given, and where in most cases the EEG became moderately or severely abnormal due to electroconvulsive therapy, changes were also noted in the response to arithmetic problems and the response to opening and closing of the eyes.

The most frequent response in the treated cases was that of a lessening of the reduction in voltage. This occurred in 16 cases. The simple problems showed greater reactivity in this lessening than the complex. This type of response is seen in figure 1.

A reversal type of reaction occurred in 6 cases. An additional 3 cases were suggestive of this type of response. By reversal, we mean the opposite of the type of response we have just mentioned. With these patients a greater reduction of the voltage occurred following

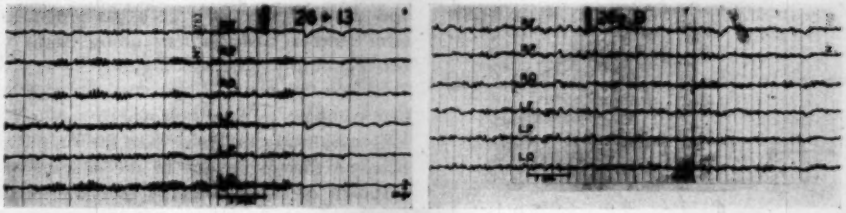


FIG. 1. On left side is EEG response to arithmetic problem solving before ECT, and to the right is EEG response after the 6th ECT.

electrically induced convulsions. This type of response is seen by looking at the right side of figure 1 first and the left side as the post-treatment situation.

With some patients there was a mixed type of response. At times they showed a reduction of blocking and at other times a reversal. Five subjects showed this mixed type of response clearly and 5 gave suggestive mixed responses. See figure 2 and table 1.

A fluctuating type of response was another category which was noted. In these instances the patient showed a markedly inconsistent response to either the simple or complex problems or both after electrically induced convul-

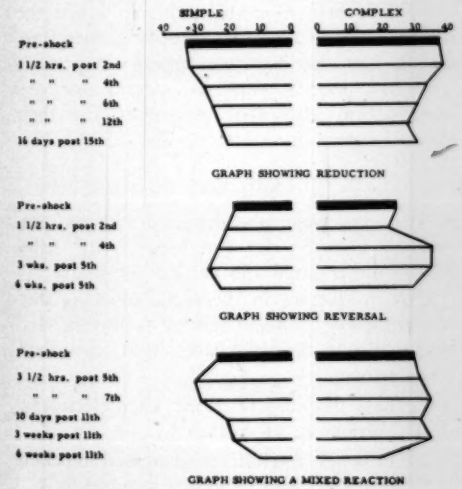
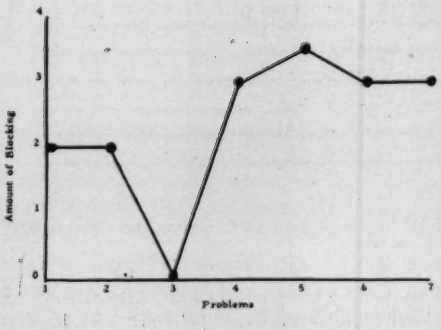


FIG. 2. Graph showing proportionate responses in alpha blocking to simple and complex problems. Note changes in blocking even after ECT has been discontinued.

TABLE 1.—GENERAL TREND OF RESPONSE OF 35 PATIENTS TO ARITHMETIC PROBLEM SOLVING FOLLOWING ELECTROCONVULSIVE THERAPY, SHOWING THE REDUCTION IN BLOCKING, INCREASED BLOCKING OR REVERSALS AND THE MIXED TYPE OF RESPONSE.

	Reduction of Blocking			*Reversal*			*Mixed*		
	T	*S*	*C*	*T*	*S*	*C*	*T*	*S*	*C*
1	X	X	X						
2				X	X	X			
3	X	X	X						
4			X				X	X	
5				X	X				X
6	X	X	X						
7	X	X	X						
8	X	X	X						
9			X				X	X	
10	X	X	X						
11	X	X	X						
12				X	X	X			
13							X	X	X
14				X	X	X			
15	X	X	X						
16				X	X	X			
17							X	X	X
18	X	X	X						
19	X	X	X						
20							X	X	X
21	X	X	X						
22						X	X	X	
23							X	X	X
24	X	X	X						
25				X	X	X			
26	X	X	X						
27				X	X				X
28	X	X	X						
29				X	X				X
30						X	X	X	
31							X	X	X
32				X	X	X			
33	X	X	X						
34	X	X	X						
35			X				X	X	

T, total; S, simple; C, complex.



GRAPH OF FLUCTUATION

FIG. 3 shows the amount of alpha blocking by one patient when given 7 arithmetic problems following the 4th ECT. Problems 1 through 3 are the simple problems, and 4 through 7 are the complex. The amount of blocking was estimated for each problem on a zero to 4 scale. This graph demonstrates the inconsistent response of blocking to simple problems. In this instance, the response to the 3rd problem showed no blocking.

sions. This can best be understood by looking at figure 3. Seven patients fitted into this group.

Other observations which were made are as follows: 1) electrically induced convulsions did not affect the giving of the correct answers, 2) no correlation was found to exist between the EEG abnormality induced by electroconvulsive therapy and the response to arithmetic problems. A change could be predicted but it was not possible to predict in what direction the change would occur. In most cases the change from the baseline went in only one direction, that is, the patient showed either a reduction of the blocking or a reversal in the blocking effect.

DISCUSSION

A psychological event, solving arithmetic problems, shows a certain neurophysiological response in the EEG. An electrically induced convulsion produces immediate psychological disturbances which are sometimes seen for considerable periods afterwards. The initial disturbance is some impairment in various aspects of the sensorium. However, these impairments and even the EEG changes after electrically induced convulsions are not correlated with improvement in psychopathology and do not give any clue as to prognosis or offer further basic understanding as to what is happening psychopathologically.

In our previous discussion we mentioned that complex problems provoked intenser and longer blocking of alpha than the simple. We believed this to be due to the amount of investment of psychic energy that was required for such a task. If sustained investment of psychic energy caused greater blocking, then less blocking of alpha would mean a decrease in the ability to invest the psychic energies in solving a problem. In the majority of cases the response to the arithmetic problems showed less blocking post electrically induced convulsions. From this orientation, electrically induced convulsions would seem to act as a sedative upon certain unpleasant psychological experiences.

In the reversal reaction the individual patient showed an increase in aplha depressing effects. We can explain this on the basis of the neurophysiological response prior to shock being inhibited and this inbihition then being released with electroconvulsions. Such a release was demonstrated by the increased blocking effect.

From what we have already described, fluctuation in the neurophysiological response also implies fluctuation in the ability to invest the psychic energies. It is not surprising, therefore, that it was this particular group of patients who had poor response to electroconvulsive therapy and also poor prognoses for their illness in general.

SUMMARY

This experiment was designed with the purpose of bridging the gap between the fields of neurophysiology and clinical psychiatry.

Certain changes in the alpha blocking during arithmetic problem solving following electroconvulsive therapy have been described. They were:

1. Less blocking of alpha after electroconvulsions in 16 cases.
2. Reversal reaction or increased blocking in 6 cases and suggestive reversal in 3.
3. Mixed reaction or combinations of the two previously mentioned reactions in 5 cases with 5 suggestive mixed reactions.

4. Fluctuating responses in 7 of the above cases.

The results suggest that the electrical changes observed reflect an alteration in the ability to invest the psychic energies. In other words this experiment would appear to show a correlation between cathectic processes and the electric activity of the cortex.

BIBLIOGRAPHY

(1) Bagchi, B. K., Howell, R. W., and Schmale, H. T.: The electroencephalographic and clinical effects of electrically induced convulsions in the treatment of mental disorders. *Am. J. Psychiat., 102:* 49-60, 1945.

(2) Chusid, J. B., and Pacella, B. L.: The EEG in the electric shock therapies. J. NERV. & MENT. DIS., *116:* 95, 1952.

(3) Cornil, L., Gastaut, H., and Ollivier, H.: Le controls electroencephalographique de l'electrochoc. *Ann. Med. Psychologique, 2:* 561, 1945.

(4) Gibbs, F. A.: Interpretation of the electroencephalogram. *J. Psychol., 4:* 365-382, 1937.

(5) Gordon, H. L.: Fifty shock therapy theories. *Mil. Surgeon, 103:* 397-401, 1948.

(6) Hetherington, R.: Psychological changes measured by a battery of tests given repeatedly. *J. Ment. Sc., 98:* 334-341, 1952.

(7) Janis, Irving L.: Psychologic effects of electric convulsive treatments. J. NERV. & MENT. DIS., *111:* 359, 1950.

(8) Kalinowsky, L. B., and Hoch, P. H.: *Shock Treatments and Other Somatic Procedures in Psychiatry,* pp. 294. New York: Grune & Stratton, Inc., 1946.

(9) Karliner, Wm.: Neurologic signs and complications of electric shock treatments: a review. J. NERV. & MENT. DIS., *107:* 1, 1948.

(10) Levy, N. A., Serota, H. M., and Grinker, R. R.: Disturbances in brain function following convulsive shock therapy; electroencephalographic and clinical studies. *Arch. Neurol. & Psychiat., 47:* 1009-1027, 1942.

(11) Lowenbach, H., and Stainbrook, E. J.: Observations on mental patients after electro-shock. *Am. J. Psychiat., 98:* 831, 1941.

(12) Moriarty, J. D., and Siemens, J. C.: Electroencephalographic study of electric shock therapy: psychotic patients treated in a U. S. Naval Hospital. *Arch Neurol. & Phychiat., 57:* 712-718, 1947.

(13) Mosovich, A., and Katzenelbogen, S.: Electroshock therapy, clinical and EEG studies. J. NERV. & MENT. DIS., *107:* 517-530, 1948.

(14) Pacella, B. L., Barrera, S. E., and Kalinowsky, L.: Variations in EEG associated with electric shock therapy of patients with mental disorders. *Arch. Neurol. & Psychiat., 47:* 367-384, 1942.

(15) Rosen, S. R., Secunda, L., and Finley, K. H. The conservative approach to use of shock therapy in mental illness. Including a study of electroencephalographic tracings before, during and after shock therapy. *Psychiat. Quart., 17:* 617-641, 1943.

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INTOXICANT DRUGS (MESCALINE AND LYSERGIC ACID DIETHYLAMIDE) IN PSYCHOTHERAPY

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Introductory Notes

In an effort to save the patient time and money, many and varied attempts have been made to shorten the course of psychoanalysis. Only those procedures which are within the realm of depth psychology deserve consideration. Among these are: 1) Steckel's method of active psychoanalysis, 2) Frank's psychocatharsis, and 3) narco-analysis. A few years ago I described another method, "deep relaxation with free ideation." This procedure makes it possible to induce in the fully awake and conscious patient, during interview in the physician's office, physical and visual experiences which may be interpreted as genuine dreams. Such experience can take the place of true dreams or can supplement incomplete or sketchy dreams. This procedure, however, cannot be used with sufficiently reliable results in every patient in whom it would appear indicated. In such cases we make use of certain drug-induced dream-like states. These are particularly effective since the patient's critical consciousness is not impaired during these states or at least remains at all times ready to intervene.

The Drug

Two synthetic substances have proved to be particularly well suited for this purpose: 1) Mescaline, an alkaloid known for many years and originally produced by Merck in Germany under the name Mescalinum sulphuricum, and 2) more recently, a newly discovered ergot derivative, d-lysergic acid diethylamide, known as LSD 25, produced by Sandoz in Switzerland. An abundance of literature on both these drugs is available in Europe as well as in the United States. However, this literature is substantially oriented toward psycho-pathology. The European psychotherapists have not used either of these two drugs, whereas in the United States they have been used to a certain extent psychotherapeutically.

I have had personal experiences with mescaline for more than seventeen years. Mescaline is administered intramuscularly in amounts varying between 0.3 and 0.5 gm. If the dose exceeds 0.3 gm., it is recommended that the injection be administered in two equal doses one hour apart. As for LSD 25, I have been using it for nearly three years in my psychiatric practice. The indications for its use, however, have become increasingly more limited with time. LSD 25 should be given in much smaller doses, namely, 30 to 60 micrograms up to a maximum of 100 micrograms; moreover, there are cases where 20 to 30 micrograms prove to be quite effective. A total of about two hundred treatments were given with these drugs.

Effect on Normals

The psychotherapeutic use of both drugs was preceded by experiments on myself as well as friends outside of my medical practice. An obvious similarity in the effects of mescaline and LSD 25 was noted during these trials: both intoxicants produce similar though more or less intensive hallucinatory states; visual and physical perceptions occurred which were most marked when the subject's eyes were closed but which also took place with the eyes open. Once their symbolism was understood, and the initial stage of the intoxication was overcome, these experiences proved to be meaningful. A certain relationship between the subject and the content of his hallucination was evident in early experiments. Now there is proof identifying the dreamer with his dreams in almost every case.

The Utilization of Drug-Induced States of Intoxication in Psychotherapy

In the course of psychotherapy, the close connection between the subject and his dream is clearly and meaningfully revealed in still another manner: the patient's essential problems are presented to him during the state of

intoxication just as they are in his dreams. This comes about in one of two ways: in some, childhood memories are uppermost, reaching very far back and with strong emotional content; instead, in others, life situations including their psychopathological history are presented in dream-like symbols. In the course of psychoanalysis this experience can be utilized and evaluated like any other material, and followed into its depth. Inasmuch as it is experienced with critical consciousness and powerful emotion, it often proves to be particularly valuable in our procedure. The fact that these experiences are artificially produced frequently proves to be, in itself, an advantage rather than a disadvantage. In our studies, it certainly did not curtail the importance of the psychotherapeutic use of the drugs. Five or six hours are required for these sessions, sometimes longer, and the investment in time must be justified by additional results. The state of intoxication is almost always associated with an apparent retardation or prolongation of treatment. Patients can often not afford this expenditure in time. Thus, they are helped if, by means of the drug effect, an emotional or memory block is broken down. Stimulation produced in this manner has one more sequel in that in almost every case it terminates in psycho-catharsis, which causes the patient to experience an exhilarating feeling of liberation. Thus, the state of intoxication proves itself to be a phase in the healing process.

Case Histories

Our experiments will be illustrated by a number of examples. I have known only one case in my entire experience in whom a single state of intoxication brought about lasting improvement. The subject, a 35-year-old printer, married for 12 years to a woman 5 years his junior, had gradually become estranged, but was unable to leave her. At first he became impotent in her presence only; later, completely. Through analysis the difficulties rooted in his childhood were mostly resolved and he felt considerably freer; impotence, however, remained unchanged. Inasmuch as time was pressing, I decided to induce a state of intoxication with 0.4 gm. of mescaline. About one hour after the injection, the patient became very cheerful and visualized, with closed eyes, a wide variety of patterns, mostly of a slightly erotic nature. In the third hour he visualized homo-erotic scenes dating back to his post-puberty period and one scene at the beach where, at the age of six, he had fallen in love for first time with a girl. Inasmuch as the results described so far were not sufficiently significant at the end of three hours, I gave the patient an additional dose of 0.1 gm. mescaline. Forty-five minutes later, this small additional dose brought about an erection; patient felt himself involved in a series of sexual experiences in which he took a very strong active part. Some of these were orgiastic scenes. From the next day on, patient's potency was fully and permanently restored.

The following example shows that mescaline-induced intoxication can be very effective even though very strongly ideational in nature:

An industrialist was once given a dose of 0.5 gm. mescaline by me at the age of 30. Fourteen years later the same individual, then a guest at my house, received a second dose. Both times, under the influence of the drug, a friendly argument took place between the patient and myself—which he knew beforehand he would lose. He fought against revealing to me memories and scenes that came to him during his state of intoxication. In the course of the first session, for the most part, sexual memories from his childhood presented themselves in symbolic fashion. These memories were merely hinted at but later were consolidated by being put into writing. In the second session, the subject of discussion was his marital problem. Gradually and despite continued resistance, the patient came to admit feelings of guilt in regard to his marital difficulties. He admitted that he gave too little love and finally ate, as a symbolic offering, a piece of cake that was lying on a plate before him. Similar symbolic expressions were produced at random. Subsequently, the subject became more easygoing and his marriage improved noticeably.

Without the two sessions and the influence of the drug, as the patient admitted, the barrier between us would not—despite our good relationship—have broken down and lasting success would not have been attained.

However, mescaline can release very strong

emotions and even states of anxiety. In this respect, mescaline is more overpowering than LSD 25 and it should be preferred to the latter in cases where the strongest possible emotional upheaval is desired. On the other hand, LSD 25 has a broader spectrum, particularly in its physical effects and lends itself to a more accurate dosage. For this reason I gradually started to use it with considerably greater frequency than mescaline; it can be used more often without the risk of harmful effects.

The importance of this was proven in the example of a 25-year-old patient who came to me with a case of almost total impotence. For a long time, analysis of his dreams, ideas and memories did not permit deep insight. We were unable to find any explanation for an excessive tactile sensitivity of the naval dating back to early childhood. A trial was made with 70 micrograms of LSD 25 during which the patient suddenly felt himself to be a small helpless child subject to titillation of the navel and genitalia and to other kinds of torture. Upon questioning his mother we found that for several months in the first half of his second year he had had much to suffer at the hands of a nurse. Photographs were presented to show how a happy, cheerful boy had, within half a year, turned into a seemingly depressed-looking child.

Despite this revelation, analytical methods did not lead much further and only after a series of 12 LSD 25 sessions during the following 15 months did the patient improve step by step. He finally was relieved of his deficiency and became fully potent. Drug-induced dreams were mostly filled with tormenting experiences in which the same childhood incidents were repeated in various guises, without much clarity. In these dreams the patient wept and wailed so vehemently that treatments could be conducted only in a hospital.

Not all patients under the influence of LSD 25, however, have the same strong urge to express themselves as the one described in this case. For this reason subsequently written statements and descriptions by the patient are important; in most cases they have a fairly good recollection of the course of events. Very often during the state of intoxication they are unable to express many things either for lack of time or because they find talking disturbing. One patient, who observed the course of his emotions particularly well, summed up five of his LSD 25-induced dreams. In each dream there reappeared only one characteristic emotion; other emotions indicated their presence but could not be brought out into the open. Also, it was impossible to foresee what the overall mood would be. The patient further referred to an experience which has been described to me by other patients as well, namely that decisive events or occurrences are preceded by a definite overall state of mind which did not, however, develop out of a situation or an object. This state of mind was always associated with a form of behavior or an expressive motion which then quasi summoned the object; this object then acquired shape either visually or by means of certain expressive motions, mostly of the hands and arms. In this connection it is made clear that it is futile for the investigator to insert himself deliberately into the course of events.

Of particular importance are those states in which birth and pre-natal experiences are re-experienced. In the case of two reliable patients it was subsequently possible to ascertain with the help of the mother that birth had been accompanied by certain difficulties, identical with those experienced during the state of intoxication. The experience of two other patients referred to the phase of cervical dilatation during labor. Two other patients relived their late embryonic stage. These latter patients both agreed that during this experience they felt that their hearts were enlarged. One of them believed that he could relate the life-threatening shock he had experienced at that time with a severe emotional shock his mother had experienced in the penultimate month of pregnancy, of which he had learned from his mother for the first time after the LSD session.

All patients were intellectuals with highly trained minds; they experienced all these phenomena spontaneously and in the absence of any suggestive influence. Thought transference cannot be taken into consideration, although it does play a somewhat considerable role in regard to the intensity and effectiveness of the LSD 25 state. This state can readily have its course guided by the patient and the physician, but it cannot be lastingly influenced.

On the other hand, the patient is sometimes able to resist very effectively the unfolding of the toxic state, even when mescaline is given.

Differentiation between Mescaline-induced and LSD 25-induced Intoxication

An outline of the difference between the behavior of the two drugs was sketched in the preceding pages. It now remains to take a somewhat closer look. The results of such a comparison are most obvious when a state of intoxication is induced in the same subject first with one drug (mescaline) and then with the other (LSD 25).

My experiment deals with a 43-year-old physician who, because of strong tensions and inhibitions, had already had analytic and similar treatment for a considerable length of time. His childhood problems had long since been revealed but he still had not undergone any decisive change. Therefore, we first tried a mescaline session and one year later, without intervening treatment, followed up with an LSD 25 session. The administration of 0.5 mg. mescaline produced a state of intoxication which was at first filled with abrupt changes from elements of utter desolation to abundance, from light to darkness. Then followed a turmoil of men, cruelty and destruction which were followed by a desert of technical standardization. Finally, alternating states, each lasting several minutes, of depressive emptiness and lightheaded euphoria followed. In the course of the year that followed, the patient became more relaxed and free, at first very rapidly, and then progressively more slowly. When this stage came to an end, we tried LSD 25 (60 μg.). The patient experienced, above all, bodily changes alternating with states of exaltation and depression. Subsequently the patient became freer even if not always in the same degree. This patient found the mescaline effect to be overpowering, deeply moving, elemental, spacious and interspersed with tensions almost schizophrenic in nature. The LSD 25 effect was described as much more circumscribed and concerned itself more with pleasurable and unpleasant sensations in which respect it resembled manic-depressive states.

Other patients expressed themselves in similar manner, mainly about the greater potency of mescaline. A direct comparison of both psychoses is possible only to a very limited degree. On the other hand, another difference exists which is expressed clearly in the psychotherapeutic use. Mescaline very seldom induces the subject to experience scenes from his personal life while LSD 25 very often works in this direction. However, this is not an iron-clad rule but rather an impression.

LSD 25 is particularly important because, following doses as low as 20μ, it can have lasting effects on organic-neurotic changes, especially in conditions associated with smooth-muscle cramps, such as chronic constipation and dysmennorrhea. Indications for these drugs should be decided upon as soon as familiarity has been acquired with the nature of the intoxicative state. The therapist must be familiar with this from his own personal experience so that he may be able to cope with the frequently somewhat difficult emotional situations arising under the influence of these drugs; he must also have experience in psychotherapy. Such experience serves to exercise caution in the case of patients afflicted with schizophrenia or endogenous depressions, thus reducing risks in such cases. I have, incidentally, never experienced any dangers of addiction.

Summary

The writer has made use of drug-induced states of intoxication as an aid in psychotherapy. To attain the states of intoxication, he has used mescaline and LSD 25. Mescaline was used in a dosage from 0.3 to 0.5 gm. intramuscularly and LSD 25 30 to 60μ administered orally. These led to states of a a dream-like nature with experiences that were clearly remembered afterwards. The procedure is indicated when it is desirable to shorten a course of therapy, reactivate a stalled treatment of a neurosis, and for the purpose of breaking down affect or memory blocks. A psychocathartic effect is almost uniformly produced. Mescaline has a more intensive effect than LSD 25 and should be preferred to the latter in cases where it is desired to obtain the strongest possible emotions. On the other hand, LSD 25 seems to have a broader spectrum; it frequently causes the patient to relive scenes from his earlier personal life and it can also have lasting influence on organic-neurotic

states. In the hands of the experienced psychotherapist, and after appropriate experimentation on oneself, and provided the indication is prudently selected, the effects of both mescaline and LSD 25 may constitute very valuable therapeutic aids.

BIBLIOGRAPHY

(1) Arnold, O. H., and Hoff, H.: Untersuchungen über die Wirkungsweise von LSD. *Wien. Ztschr. Nervenh. 6:* 129, 1953.

(2) Arnold, O. H., and Hoff, H.: Körperschemastörungen bei LSD 25. *Wien. Ztschr. Nervenh. 6:* 259, 1953.

(3) Beringer, K.: *Der Meskalinrausch.* Berlin: Springer-Verlag, 1927.

(4) Busch, A. K., and Johnson, W. C.: LSD 25 as an aid in psychotherapy (preliminary report of a new drug). *Dis. Nerv. Syst., 11:* 241, 1950.

(5) Frederking, W.: Ueber die Tiefenentspannung und das Bildern. *Psyche 2:* 211, 1949.

(6) Frederking, W.: Ueber die Verwendung von Rauschdrogen (Meskalin und Lysergsäurediaethylamid) in der Psychotherapie. *Psyche, 7:* 342, 1953/54.

(7) Forber, G. R., and Goldner, R. D.: Experimental psychological studies with lysergic acid diethylamide (LSD 25). *Arch. Neurol. (Am.), 65:* 581, 1951.

(8) Hoch, P. H., Cattell, J. P., and Pennes, H. H.: Effects of mescaline and lysergic acid (d-LSD-25). *Am. J. Psychiat., 108:* 579, 1952.

(9) Katzenelbogen, S., and Ai Ding Fang: Narcosynthesis effects of sodium amytal, methedrine and LSD-25. *Dis. Nerv. Syst., 14:* 85, 1953.

(10) Matefi, L.: Mezcalin- und Lysergsäurediaethylamid-Rausch. Selbstversuche mit besonderer Berücksichtigung eines Zeichentests. *Confinia neurol., 12:* 146, 1952.

(11) Mayer-Gross, W.: Experimental psychoses and other mental abnormalities produced by drugs. *Brit. M. J.,* 317, 1951.

(12) Mayer-Gross, W., McAdam, W., and Walker, J. W.: Psychological and biological effects of lysergic acid diethylamide. *Nature, 168:* 827, 1951.

(13) Rinkel, M., De Shon, H. J., Hyde R. W., and Solomon, H. C.: Experimental schizophrenia-like symptoms. *Am. J. Psychiat., 108:* 572, 1952.

(14) Savage, C.: Lysergic acid diethylamide (LSD 25), a clinical phychological study. *Am. J. Psychiat. 108:* 896, 1952.

(15) De Shon, H. J., Rinkel, M., and Solomon, H. C.: Mental changes experimentally produced by L.S.D. (d-Lysergic acid diethylamide tartrate). *Psychiat. Quart., 26:* 33, 1952.

(16) Stoll, W. A.: Lysergsäure-diäthylamid, ein Phantastikum aus der Mutterkorngruppe. *Schweiz. Arch. Neurol., 60:* 279, 1947.

(17) Stoll, W. A.: Rorschach-Versuche unter Lysergsäure-Diaethylamid-Wirkung. *Rorschachiana, 1:* 249, 1952.

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SUDDEN DEATH IN INSULIN COMA TREATMENT FOR SCHIZOPHRENIA

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The following case is of considerable interest as, although death in insulin coma treatment is a recognized hazard, the majority of such deaths occur during the phase of prolonged or irreversible coma some hours or days, or in a few cases weeks, after the attempted interruption of the coma. In this case death occurred 15 minutes after the administration by nasal catheter of 20 oz. of 33 1/3% glucose and before the onset of irreversible coma could be said to exist. This is the only death in 247 cases treated at Barrow Hospital in the four years ending September 1952 by the insulin coma method.

Case R. G., aged 40, salesman. Physically he was a well-built, thick-set man. He was a case of katotonic schizophrenia. His first mental illness occurred in 1947 when he was a patient in a Glasgow mental hospital for four months, with an early schizophrenic episode. He did not receive insulin treatment. He returned to his work as a salesman and managed to carry on until two months before his admission to Barrow Hospital as a voluntary patient on August 23, 1950. He expressed delusions of being influenced by external forces, his conversation was vague and diffuse, and obsessional traits were present. Episodes of mutism and refusal of food occurred. Apart from a dysrhythmic E.E.G. record, all laboratory investigations were normal and x-ray of chest was normal. On September 28, 1950 he commenced insulin treatment, during the course of which he improved greatly and developed good insight. After 23 treatments and 13 comas, on a dosage of around 160 units of insulin, he developed a severe tracheo-bronchitis and treatment was terminated on October 23, 1950. No fits occurred during the treatment. Duration of coma was usually 30 minutes.

On recovery from the bronchitis he was unwilling to resume treatment and pressed for his discharge. As he was by then free from mood disturbance and delusional ideas and had good insight, he was discharged on November 11, 1950.

On January 3, 1952, he was readmitted in a katotonic state in which he alternated between noisy aggressive outbursts and periods of mutism. He had done only three months' work in the intervening period.

On May 6, 1952 he commenced a course of insulin treatment commencing with 16 units of insulin and increased in steps of 16. Coma dose was about 160 units. This was interrupted on May 23, 1952 after five comas, because he had no upper teeth and his lower teeth were cutting his upper gums while in coma. Treatment was resumed on June 26, 1952 after extraction of the teeth. Meanwhile his mental condition had improved somewhat. No bronchitis had occurred during this hospitalization. On the fourth coma day one fit occurred during the coma stage. On July 18, 1952 he received his 35th treatment and 20th coma with 160 units of insulin. At 10.30 a.m., 7 oz. of glucose were administered by nasal catheter. At 10:40 a.m., it was noticed by the doctor on duty that his color was good and the appearances were normal. At 10:44 or 10:45 a.m., it was seen that he had become very cyanosed and breathing was not apparent. All attempts at resusciation, including nikethamide, oxygen and artificial respiration, were without avail. On post-morten examination the only abnormalities found were deep cyanosis of the head and neck, the lungs were deeply congested, the chambers of the right side of the heart wereacutely dilated and the liver and the kidneys were congested. The brain also was deeply congested but no gross abnormality was detected by the naked eye.

In the opinion of the pathologist, Dr. A. L. Taylor, the cause of death was cerebral anoxia and respiratory failure following insulin shock therapy, and no specific brain changes, other than those of cerebral anoxia, were seen on microscopic examination.

Discussion

With improved technique in insulin coma treatment in the therapy of schizophrenia the mortality rate has diminished considerably since its introduction by Sakel in 1933. Deaths in insulin coma occur in two ways: 1) sudden and unexpected death in a coma phase itself or very soon after interruption of the coma, and 2) death in prolonged or irreversible hypoglycemic coma. The latter is much the commoner type of fatality.

A number of deaths have been reported in the course of modified or subcoma insulin as well as in coma insulin treatment.

Mortality figures have varied considerably, but the incidence over all is low in relation to the value of the treatment, and is certainly considerably less than one per cent. In this hospital there has been one death in a series of 247 cases.

Sargent and Slater (9) report nil mortality in 400 cases in eight years. Paster and Holtzman (6) in a series of 241 cases, had no deaths. Malamud (5) quotes D. J. Sullivan as having one death in 322 cases treated by subcoma insulin. This death occurred after 60 hours of irreversible coma with terminal broncho-pneumonia. In this connection he refers to the studies of Rivers and Rome (7) whose conclusions was that neither the size of the initial dose, the total dosage, the depth of the coma nor the time elapsing between the onset of coma and the institution of glucose therapy seemed to play a role, but that it was a problem of some individual factor such as hypersensitivity, a conclusion with which Malamud agreed. This view is not usually accepted, however; for example, the investigations of Spencer (11) led him to the conclusion that if prolonged coma is to develop, it tends to develop early in the treatment, and especially if the coma dose is reached early—i.e., by working up to the coma dose too rapidly—especially if reached before the tenth day of treatment. He believes that prolonged coma is due to the occurrence of irreversible changes in the brain, or part of it) and that post-coma confusion is a warning sign that the patient may develop prolonged coma the next day, and that such patients should be given a few days' rest from treatment.

W. Sargent (8) reporting on seven years' experience of modified (sub coma) insulin treatment of about 1000 cases reported no fatal cases. Scheflen et al. (10), in an interesting study of seven cases of death under insulin coma treatment, and one in subcoma insulin treatment which occurred in the region covered by Massachusetts Department of Mental Health, in the 11 years from 1941-1951 (inclusive) records that five deaths occurred in irreversible coma, some hours or days after attempted interruption. The other three died suddenly and unexpectedly: one ten minutes after onset of coma, one five minutes before the scheduled time of interruption, and one ten minutes after attempted interruption of coma. The case I am reporting has some features in common with the last case described by Scheflen, who reports that an uneventful coma of one hour's duration occurred with 450 units of insulin.

"Termination was attempted with intravenous administration of 37 gm. glucose. Ten minutes later 13 gm. more was given. At this time, during the insertion of a Levin tube, the patient suddenly became cyanotic and pulseless. Resusciation measures were of no avail." The dosage of insulin used in some of the cases described would appear to many people to be very unusual. For example case no. 7 is said to have had as successive doses: 50, 100, 200, 400, 800, 1600, 1600, 1600, 2000, and on the tenth day a dose of 500 units which proved fatal.

Jetter and Schelflen (3) report that the neuropathological picture of fatal hypoglycemia is not distinguishable from that of anoxia caused by other agents, and he agrees with similar findings by L. R. Morrison (1946).

Himwich (2) also emphasizes the similarity between the symptoms of progressive hypoglycemia and acute anoxia. Recent work by Lovatt Doust and Schneider (1) is of considerable interest in this respect. They carried out a spectroscopic oximetric study of arterial blood saturation levels in 13 patients undergoing insulin coma therapy. The measurements were taken every five minutes over the course of eight to ten hours in each patient, and coma was reached in 12 out of 13 patients. A progressive and profound anoxemia developed, which reached its lowest level in the coma phase, with a rapid rise to above normal after interruption of coma by glucose. It is tempting to accept the view that has been put forward that those deaths which occur in or soon after the coma stage are the result of a progressive anoxic process (which is according to Himwich due to the absence of the glucose substrate so that such oxygen as is available cannot be utilized). It is assumed that this progressive anoxic process goes too far and an irreversible change in the cerebral cellular microbiochemical exchanges takes place leading to failure of the vital centers, the respiratory center failing first and then the circulatory center. This is not the whole explanation, however, since the level of consciousness does not run parallel with the level of glucose in the blood in many cases. This is well demonstrated by one case of the writer who was extremely sensitive to insulin. A test dose of 7 units depressed his blood sugar to zero on this occasion, associated with de-

layed return to normal, but although there was a definite impairment of consciousness he did not develop coma. It is also possible, however, that the effect of the anoxic process linked with the hypoglycemia may exert marked effects elsewhere in the body, and particularly on the heart muscle which is very sensitive to oxygen lack, and that the causes of actual death may be extra-cerebral: and it will be remembered that, in the case recorded here, the chambers of the right side of the heart were acutely dilated. Lovatt Doust and Schneider (1) have shown how closely parallel the differentials of consciousness are with the blood oxygen saturation level when measured oximetrically, both in natural sleep and in hypoglycemia, and important clues to the pathology of hypoglycemic encephalopathy may well be obtained by the study of the relation between blood sugar concentration and the blood oxygen saturation in cases of irreversible coma occurring during treatment. In fatal cases also it would seem to be important that a widespread detailed examination of organs should be made; this may well reveal important facts, and possibly that the actual cause of death is extracerebral and not intracerebral.

Summary

A case of sudden death during insulin coma treatment, 15 minutes after the administration of a nasal feed to interrupt the coma, is described. The incidence of death occurring during insulin treatment of mental disorders, and the likely mechanisms involved are discussed, including the possibility that the cause of death may be extracerebral, such as cardiac failure due to an anoxic process.

My thanks are due to Dr. A. L. Taylor, pathologist, for the post-mortem findings, and to Dr. R. E. Hemphill, Medical Superintendent, Bristol Mental Hospitals, for his interest in this paper.

References

(1) Doust, Lovatt and Schneider: *Brit. M. J., 1:* 499, 1952.
(2) Himwich, H. E.: *Brain Metabolism and Cerebral Disorders.* Baltimore: The Williams & Wilkins Co., 1951.
(3) Jetter and Scheflen: Delayed pathologic manifestation of hypoglycemic coma. *Jr. Neuropath & Exper. Neurol., 9:* 317, July 1952.
(4) Malamud, N.: Fatalities resulting from treatment with subshock doses of insulin. *Am. J. Psychiat., 105:* 373, Nov. 1948.
(5) Malamud, N.: *Am. J. Psychiat., 105:* 703, 1949.
(6) Paster and Holtzman: A study of 1,000 psychotic veterans treated with insulin and electric shock. *Am. J. Psychiat., 105:* 811, 1949.
(7) Rivers and Rome: Prolonged insulin coma treatment of schizophrenia. *Arch. Neurol. & Psychiat., 51:* 550, June 1944.
(8) Sargant, Wm.: Seven years' experience with modified insulin treatment in neuroses and psychoses. *Am. J. Psychiat., 105:* 821, 1949.
(9) Sargant and Slater: *Physical Methods of Treatment in Psychiatry,* 2nd ed. Baltimore: The Williams & Wilkins Co., 1948.
(10) Scheflin, Reiner, and Jetter: *Arch. Neurol. & Psychiat., 67:* 32, June 1952.
(11) Spencer, A. M.: Post hypoglycemic encephalopathy. *Jr. Ment. Sc., 44:* 396, July 1948.

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CHARACTERISTIC HEADACHE RESULTING FROM PROLONGED USE OF ERGOT DERIVATIVES

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The cases reported in this article should serve to warn the physician who places ergot derivatives (gynergen, dihydroergotamine, cafergot, etc.) in the hands of migraine patients for self-administration. After twenty-five years of observation in handling migraine cases I have formed the following conclusions:

1. Prolonged use of ergot derivatives in any appreciable amount results in increased frequency of migraine attacks. Eventually the headaches begin to occur daily. At the same time, there is increased reliance upon the ergot preparation for relief, leading in some cases to total addiction.

2. A *second* type of headache which the patients call "a gynergen headache," or "an ergotamine headache," very often appears *in addition to* the migraine headache. The patients can differentiate clearly between the two types of headache. The ergotamine headache is characterized by a dull, constant occipital pressure which may spread along both parietal bones in the form of a bonnet, or cap. This type of headache appears to act as a trigger, bringing on, and gradually merging into, the typical, one-sided, throbbing migraine headache.

In cases where there is ergot addiction, even after the migraine headaches no longer appear, the ergotamine headache persists daily and can be relieved only by repeated injections of the ergot derivative.

As the patient comes to rely upon more and increased doses of ergotamine to control the headache attacks he may begin to suffer from chronic nausea which persists continuously between headache attacks.

Since 1931 I have been using the two steroids, alpha-estradiol benzoate and testosterone propionate as a preventive treatment for migraine. I have found that it is possible to break the ergot habituation with steroid treatment. However, the patient undergoes a difficult two or three weeks during the withdrawal period. For from one to three weeks after withdrawal of the ergot derivative and under steroid treatment they still retain the daily occipital headache in extremely severe form. The migraine headaches occur much less frequently and severally during this period. However, it appears that the steroids "cushion" the shock of withdrawal, which otherwise proves dangerous and severe (see Case 2 below).

The following case is the only one in which I have been unable to break the ergot habituation.

Case 1: Miss E. S. Single. Age 37 years. First seen September 17, 1937. History: migraine headaches from the age of 5. From age 28 to 30 headaches became more frequent and severe, occurring weekly and lasting 2 to 4 days. In 1935 an internist had prescribed gynergen for self-administration, starting with 0.5 mg. weekly. Shortly thereafter the headaches began to occur every other day, and a few weeks later, daily. In 1936 she had both meningeal arteries tied, with no relief. When I first saw her she was using two 0.5 mg. ampoules daily to control headache attacks. She was emaciated, exhausted, and complained of pain, numbness and tingling of the legs, with tenderness over the calf muscles—all typical symptoms of ergotism.

Upon questioning she explained that she had two kinds of headache: 1) a headache which she associated with *lack* of ergotamine, and 2) her typical familiar migraine headache. At my request Miss S. wrote the following description of her "gynergen headache":

"Since taking ergotamine tartrate hypodermically over a period of three years, the symptoms are nothing like the old migraine symptoms. Now I usually feel an inexplicable sensation in the pit of my stomach, extreme nervousness, dimmed eyesight . . . if I do not take the hypo, pain—not on one side of the head as formerly—but mostly in the back of the head at the base of the brain, appears and gets worse. If I do get the drug in time, I will invariably get relief in a short time, except during my menstrual period, and shortly before and after."

She stated that when the migraine headache occurred just before her monthly period, it did not yield to ergotamine, and she was forced to supplement the daily injections with codein and other drugs, and even then was unable to completely alleviate the migraine headaches. She stated that the head-

ache at the time of the period consisted both of a dull, constant occipital pain, as on ordinary days, plus the one-sided temporal and eye pain which she associated with true migraine.

In 1937 and 1938 this patient was treated with alpha-estradiol benzoate which satisfactorily relieved her one-sided migraine headache. However, she was unable to stop the use of ergotamine tartrate twice daily because otherwise she found the occipital headache unbearable. In 1938 she discontinued treatment and the migraine headaches began to recur. From 1939 she informed me of her progress by mail. Her prostration and the pains in the legs slowly became worse and she was finally confined to the house.

In 1944 and 1945 she entered two different institutions where attempts at withdrawal of ergotamine tartrate were attempted. Both attempts failed.

In October, 1946 I visited the patient in her home to have a complete history stenotyped. She was decrepit, unable to walk except for a few hobbling steps. She was emaciated and exhausted. She stated the headaches were the same; ergotamine tartrate controlled the daily occipital headache, but every 7 to 10 days she had an additional, severer, one-sided headache which ergotamine tartrate plus codein and other drugs failed to relieve.

October 4, 1948 she reported to me by telephone: She stated that in the last year, her 48th, the migraine headache had disappeared. (Note: the disappearance of migraine headaches around the 48th year is common.) She reported that she was "better," though her legs still bothered her. She could walk more and was able to leave the house occasionally. She stated, however, that she still had to take ½ ampoule of gynergen twice daily in order to control the continuing occipital headache.

In January, 1952, this patient made the front pages of the local newspapers. She attempted to commit suicide.

Case 2: Mrs. E. R. Aged 60. First seen February 26, 1943. History: Her migraine headaches first appeared at age 26, after the birth of her second child. They occurred once per month, sometimes preceding her monthly period; more often they occurred at the end of the period, lasting from two to three days. The headaches occurred on either one side of the head or the other; there was always some occipital pressure with the attacks.

By 1940 the attacks had begun to recur twice a month. She consulted several doctors and went to several clinics. Gynergen was prescribed for self-administration. The headaches began to increase in frequency until by 1942 they occurred every other day.

When I first saw this patient in 1943 she stated that for the past five months she had had daily headaches. She further stated that she had two distinctly different headaches: 1) an occasional one-sided migraine headache, and 2) a daily, occipital headache.

Before beginning the steroid treatment, and without informing me of her intention, she decided on her own to quit gynergen, and discontinued her daily dose for the first time in years. This sudden withdrawal was followed by swelling of face, lips, arms, and legs, her face turned black, and in her own words, "for twenty-four hours she felt as if she were going to die." Upon institution of alpha-estradiol benzoate injections these symptoms disappeared. After a week the daily occipital headache disappeared; the migraine headaches continued to recur at much rarer intervals.

Case 3: Mrs. P. L., schoolteacher. Age 63. History: Menopause at 47; sub-total hysterectomy in 1951. Severe half-sided migraine headaches began in 1929, in her 30th year. In 1938 or 1939 she was given gynergen for self-administration, later ergotrate was substituted for gynergen. Thereafter her headaches began to recur more frequently.

On July 5, 1952, she wrote me for an appointment, saying, "For the past several years I have been awakened with headaches about four nights in six. I had to take six months' leave, as I was unable to perform my duties."

I saw this patient August 4, 1952, and began steroid treatment. Within a week she was able to get along with two weekly injections of ergotrate. She returned to her home in another city, continuing the treatment, and reporting by mail.

January 14, 1953: "I am still holding my good record of not missing a day at school this semester."

September 16, 1953: "I went through last year with only a few days' loss of time from my job."

Owing to the nature of her work this patient was never able to completely discontinue the occasional injections of ergotrate which she took when the occipital headache was severe. However, she reported that her migraine headaches had disappeared.

Case 4: Mrs. V. N. Age 48. History: Menopause at 46. Had migraine headaches from early childhood which occurred weekly for 24-hour periods. Age 46, migraine attacks became extremely severe, lasting from two to four days weekly. In 1950 she received dyhydroergotamine for self-administration. Headaches increased in frequency. Started cafergot in 1952 (summer.) Headaches increased to four or five attacks weekly.

I first saw this patient October 27, 1952. She was complaining of daily headaches and constant nausea. She was then taking from three to five tablets of cafergot daily in order to control the headaches.

Two days after steroid treatment was instituted she was able to throw away her cafergot. The persistent nausea disappeared. Her headaches gradually diminished in frequency until they occurred once each month, lasting for about four hours.

Case 5: Mrs. C. C. Age 35. History: Migraine headaches since 7 years of age. Headaches persisted during pregnancy in 32nd year. After third month of pregnancy, headaches became so severe that doctor came every morning to administer gynergen hypodermically. After her child was born she administered gynergen herself. At first two or three injections per week were sufficient, but as headaches increased in frequency, she began finding it necessary to take 4 to 5 injections weekly. At age 35 she was

having severe daily headaches, which necessitated daily injections.

This patient was first seen in March, 1940. She complained of having two types of headaches: 1) a daily headache with dull, constant occipital pressure, which responded to 0.5 mg. gynergen; 2) a half-sided throbbing migraine headache which occurred about once weekly, and which was only partially relieved by 1 cc. of gynergen. After a month of steroid treatment and withdrawal of gynergen the posterior headache disappeared, and the true migraine headache persisted about once a week, in milder form.

Mrs. C. wrote the following description of the effect of gynergen injections:

"After taking gynergen, the under part of my nose becomes black; I look as though I have a dirty face, and my entire body looks as if badly sunburned; it's a sort of purple color. This remains all day."

Case 6: Miss M. R. Age 48. History: Both parents had migraine headaches. Miss R. had migraine headaches from early childhood. In 1937 the headaches became extremely severe, occurring twice weekly. In that year she was given gynergen for self-administration. The headaches began to appear daily.

I first saw Miss R. in May, 1953. She reported that she was forced to get up at 5 o'clock each morning to take an injection of gynergen in order to abort the headache which was then beginning. She stated that she had felt for some time that her daily headaches were "different" from the migraine attacks she had had all her life, and that she attributed the daily dull, constant, occipital headache directly to the use of gynergen. She still had an occasional migraine headache, one-sided and thumping, but that they were occurring only about every two weeks.

Miss R. returned to my office August 30, 1954. She still complains of daily "gynergen" headaches which necessitate early morning injections. She exhibits the following signs of ergotism: the skin beneath her nose is mottled and purplish; her face is dusky, and the body skin is purplish bronze. As soon as her present appointive job in another city expires she plans to come in for steroid treatment in order to withdraw the gynergen. At present she holds a highly responsible position, and states that she is utterly dependent upon the gynergen in order to get her work accomplished.

Case 7: Miss V. S. Age 48. History: Migraine headaches since 16 years of age. At age 31, after an appendectomy, the headaches became more frequent, occurring 4 or 5 times monthly. At age 36, dihydroergotamine was prescribed for self-administration. Soon thereafter the headaches increased in frequency.

When first seen in April, 1954, this patient complained of daily headaches which necessitated daily use of d.h.e. Upon questioning she described the daily headache as a dull, constant, posterior headache which, if not checked, merged into an extremely violent, thumping, one-sided migraine headache.

Steroid injections were begun. Miss S. returned to her home city, continuing treatment and reporting by mail. She had an extremely difficult time for some three weeks, during which she reported by mail that "she thought she would go crazy with the pain." By July the daily headaches were comparatively mild.

On August 14 she wrote: "I have had no severe migraine headaches since July 4. The secondary, or ergot (posterior) headache now yields to black coffee and benzedrine. I am much encouraged, because I feel so much better generally."

Case 8: Mr. R. B. Age 43. History: Migraine headaches since age 20. At age 38, headaches became very severe. Gynergen was prescribed for self-administration.

I saw this patient in May, 1953, at which time he complained of a daily constant, dull, posterior headache which he found unbearable unless he took 1 cc. gynergen early in the morning.

After steroid treatment his daily headaches began to disappear.

June 3, 1953, he wrote: "It looks as though I am cured, as I haven't had a headache since arriving home (8 days.)"

September 14, 1954: "As far as I am concerned I have been getting along fine considering the hours I keep and work. After I quit having headaches I went on a maintenance dose . . ."

Case 9: Mrs. A. R. I first saw this patient in May, 1951. Since she has done quite a bit of writing, and has an unusual gift of description, and is most articulate, I recently asked her to write her own account of her experiences with migraine treatment. The following was received July 19, 1954:

"I suppose I could start this by saying that I am fifty years old and from the time I was nine, I can remember having migraine headaches. As a child they lasted twenty-four hours and during this time I was very nauseated as well. As I grew older they continued in much the same pattern; they came once or twice a month, usually before my period and after or nearly always when I was anticipating something pleasant or otherwise.

"After I was married and had my child at the end of the first year of marriage the headaches became much worse and from then on I had them more often.

"About 1939—the early part—I had a hysterectomy. Sometime after this, perhaps within the same year, my doctor got one of the ergotrate preparations for me to try for the migraines which continued and were as always severe. These little white tablets would make me very ill and dizzy for about an hour or two, during which time I had to stay in bed, but after that the headache would leave and I would be over the ordeal for a while. I think I took these tablets over a period of four or five years, having to increase the dose as time went on in order to get the same result.

"At some time after that dihydroergotamine swam into my ken and this I began to use with a hypodermic. I had used the injection for taking the ergotamine for a while before this, too, so I was used to the procedure.

"The injection was a much better way of getting results, for it worked more quickly and I felt ill

from the medicine less long a time. But over the years, the headaches continued, and I began to feel that they were becoming more frequent and that I was having to increase the injections to keep from having more and more headaches. I also felt that I was having two types of headache—one which seemed to be the migraine and one which I felt was due to the drug.

"By 1950 I was frantic as I was taking an injection a day and, if I had anything to do in the evening or felt I should be at all normal for my family, I had to take another injection toward evening. I felt horrible all the time even when the migraine was checked. I kept asking my doctor if I was not taking much too much and if I wasn't having a reaction from the drug, as well as the migraine. I would be told by each doctor that I would not have the migraine forever and that it would just all stop some day—rather by magic, I inferred, though my reason made this seem unrealistic!"

It was at this time that Mrs. R. appeared in my office, and steroid treatment was begun. She describes the results as follows:

" . . . I was more than happy to stop the dihydroergotamine injections which I'd been giving to myself, and found it only a relief not to have to rise groggily each morning about four or five o'clock and force myself through the ordeal of getting an injection so I could go back to bed for a few hours in order to have a little time during the middle of the day when I could get about and do my work before I began to feel ill and miserable again!

"Soon after my (steroid) treatment started, I was able to see that the odd second headache was beginning to leave me and when I could once more discern that I had left it behind me, it was a great relief to find that the migraine—which I still had, though much less often—no longer carried the nausea with it."

Summary

In many cases where ergot derivatives are prescribed for self-administration in migraine, ergot addiction will result because the headaches increase in frequency, necessitating more frequent use of the ergot preparation. I believe that the danger signal of addiction sounds when the patient develops a type of headache differentiated from the true migraine headache in that it is a constant, dull, posterior (occipital) type of pain. So far as I know, steroid treatment is the only method by which such ergot addiction is stopped.

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PERSISTENT SENSORY SYMPTOMS FOLLOWING SPONTANEOUS HYPOGLYCEMIA: RESPONSE TO CHLORPROMAZINE

Report of a case

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Although the occurrence of permanent neurologic damage following a prolonged hypoglycemic attack has recently been stressed (2), Baker was of the opinion that damage to the nervous system giving rise to prolonged or permanent functional impairment had been insufficiently emphasized and was commoner than the sporadic reports of cases appeared to indicate (1). Most of the sequelae described have taken the form of mental deterioration (2, 8, 18), though focal neurologic disturbances (2, 6) and lower motor neurone lesions either with or without accompanying mental changes (12, 14) have also been described. Disorders of sensation, although rarely reported, may also be a sequel both of a spontaneous or of an induced hypoglycemic attack. Though usually transitory, sensory symptoms of a distressing nature may, on occasions, be a permanent sequel. An example of this is illustrated by the following case.

Case Report

Mrs. M. S., a 49-year-old housewife, was referred for psychiatric opinion in June 1953, on account of persistent disagreeable numbness of both hands. No other cause having been discovered it was thought that there might be an emotional basis for this complaint.

Family history. Her father, a stable person, died aged 59 years of tuberculosis. Her mother was alive, aged 76 years, having survived an operation for carcinoma of the colon. Of 13 siblings, 2 died of tuberculosis, and one of lead-poisoning, while four others died in infancy. The remaining 6 were alive and well. There was no family history of mental disease.

Personal history. Up to the time of her illness the patient's life had been uneventful. Her childhood and development were apparently normal and apart from pneumonia at the age of 17 she had suffered no serious illness. She had been married happily since the age of 20 and had 3 children, a son aged 26 and two daughters aged 25 and 22 years, the eldest of whom had become depressed while nursing her mother in 1952, but recovered after some months following treatment with electro-convulsion therapy at another clinic.

History of illness. During 1948 the patient started to have attacks characterized by a short period of restless behavior followed by loss of consciousness initially of up to 2 hours' duration. On some occasions the alteration in consciousness was less profound and she would wander about in a dazed fashion, remembering nothing on recovery. These attacks occurred at monthly intervals, usually at the time of menstruation. On investigation no clear evidence of physical disease was discovered; X-ray of the skull and chest, a Wasserman test, and C.S.F. examination all gave negative results. An E.E.G., however, revealed a marked general dysrhythmia suggestive of epilepsy, though no focal disturbance. Accordingly a tentative diagnosis of epilepsy was made.

Her attacks continued with increasing severity until 1951, when she was admitted to hospital following an attack ushered in by a generalised epileptic convulsion and succeeded by a deepening depression of all voluntary activity. She remained in a stuporous condition for 6 days, and it was on recovery from this that she first complained of a feeling of numbness in both hands.

Over the next few months she had a further series of attacks of up to 8 hours' duration, and in addition to persistent numbness of the hands complained of weakness of the legs and loss of energy.

In April 1952 her physician formed the opinion that her attacks might be the result of spontaneous hypoglycemia. This was confirmed by an abnormal glucose tolerance test and the finding of casual blood-sugar levels ranging from 35 to 70 mg. per cent. As a result an exploratory laparotomy was performed, and a typical islet-cell adenoma discovered and removed from the head of the pancreas. Following this operation her attacks completely ceased and her blood sugar level returned to normal (100 to 150 mg. per cent).

Despite this the numbness in her hands continued unchanged and, because physiotherapy and other symptomatic treatment failed to give relief, in May 1953 a further investigation was carried out—though once again with negative results. Although X-ray of her shoulder girdle showed both 7th cervical transverse processes to be abnormally long, there was no evidence of cervical rib or other lesion compressing the neuro-vascular bundle. It was therefore considered likely that the symptom was of psychogenic origin and possible that her mental state might have

been impaired by the previously recurring and prolonged hypoglycemic episodes.

On examination (June 1953). Apart from moderate depression and emotional lability her demeanor was normal. Her only complaint was of a constant disagreeable sensation in both hands which she described as numbness or alternatively as an electric sensation. This, which affected the whole of both hands and all digits, extended up to, though not above, wrist level. The sensation never left her, was exacerbated by cold and by using her hands, and only partially relieved by warmth. She found the constancy of the symptom more intolerable than its severity. Her discomfort was not relieved by the usual analgesics and seemed to bear little relation to her emotional state, or to environmetally adverse factors. Apart from a tendency to drop her needle while sewing, and crockery on odd occasions, she complained of no motor disability.

On physical examination there were no gross signs of nervous disorder. Her sense of smell, though imperfect, was preserved, and no cranial nerve abnormalities were elicited. The motor system was normal, and all deep and superficial reflexes present and equal. On detailed sensory testing there was no impairment of light touch, pin-prick, or position sense in either hand or elsewhere. She was able to identify correctly a number of small commonplace objects placed unseen within her grasp. Palm-writing was however poorly recognised and there was some, though slight, impairment of two-point discrimination at all finger-tips.

The skin of both hands was normal in texture, though both were red, congested and markedly cold to the touch even during warm weather. (Reactive hyperemia 3 secs.) Both radial pulses were normal on palpation and qualitatively unaltered by downward traction or other positioning of her arms.

No abnormalities were discovered in any other system, apart from mild hypertension (B.P. 170/105). Because of mild hot flushes she was thought to be menopausal, though menstruation was not irregular when first examined.

Formal mental testing at this juncture and subsequently showed no evidence of organic mental impairment, or of any psychiatric disturbance apart from depression and anxiety secondary and proportionate to her physical disability.

Investigations. R.B.C., 4.46; Hemoglobin, 88 per cent (13.0 gm./100 cc); C.I., 0.99; W.B.C., 7,500 cu.mm, normal differential; E.M.G., no apparent abnormality of the intrinsic hand muscles. Faradic stimulation of the 1st dorsal interossei elicited a normal response: KCC>ACC. Stimulation of the median nerve at the wrist also gave a normal result.

Psychological tests. The Wechsler-Bellevue results indicated average intelligence, the deterioration index being insignificant; the Bender-Gestalt a normal result with some simplification of designs. The Rorschach record had an organic flavor, though this was indefinite, several important organic signs, including color-naming, being absent.

Course. The patient was seen at the clinic by the author at intervals of 3 to 4 weeks over the subsequent year, during which time she was treated empirically with a vitamin B-complex preparation and other symptomatic remedies. There was absolutely no response to treatment, and her discomfort continued unabated, fluctuating only slightly according to the temperature. Although considerably depressed at times by the persistence of her symptoms, she never gave up hope of their ultimate alleviation and, according to her husband, endeavored to live as normal a life as possible. Finally in May 1954 she was treated experimentally with Chlorpromazine, 75 mg. daily. After two weeks the dose was increased to 150 mg. daily, there being no side-effects. The response to this treatment was interesting. She stated that although the condition of her hands felt unchanged she found her discomfort much more bearable, and that altogether she felt very much better in herself. Objectively there was a striking change in her demeanor, which was noticeable as soon as she entered the clinic. Not only did she appear gayer and brisker in her movements, but was now no longer likely to dissolve into tears when discussing her symptoms. Two weeks later treatment was withdrawn and within a few days she had relapsed into her previously depressed and complaining state. However, on re-starting treatment her condition improved to the same extent as before, which improvement has been maintained.

Comment

The absence of any definite objective findings made the assessment of the patient's complaint difficult. Originally her symptoms were regarded as having an emotional basis, and not a result of organic nervous disease. However, prolonged scrutiny of the patient's personality and life situation, together with projective and other psychological tests revealed nothing to suggest that her depression and emotionality were other than a reaction to constant nagging discomfort.

It is also unlikely that the menopause contributed to this patient's complaint, for not only was the numbness in her hands present for almost three years before any disturbance of menstruation but, when this did occur, there was no exacerbation of her condition, nor did fresh symptoms appear, other than mild hot flushes of negligible intensity. Futhermore the resemblance of some symptoms of hypoglycemia to those of the climacteric has been mentioned by Wilder (19) who observed acroparasthesiae, depression and irritability in some of his cases.

The sudden onset of the symptoms and the absence of certain physical signs appeared to

exclude lesions resulting from local interference with the nerve supply to the hands or due to pressure on the neurovascular bundle. Despite the fact that both 7th cervical transverse processes were longer than normal, there was no evidence to suggest that the complaint was due to acroparasthesiae resulting from the rib-pressure syndrome (16) nor was the lack of thenar atrophy or the distribution of the pain characteristic of bilateral compression of the median nerves in the carpal tunnel (4).

By and large the accumulated evidence regarding this case points to hypoglycemia as a cause of the symptoms, though both the nature of the occurrence and the unremitting persistence of the complaint is difficult to explain. This is strongly supported by the fact that the sensory disorder appeared suddenly on recovery from the most severe and prolonged of all the patient's hypoglycemic attacks.

Symptoms of the same or of a similar kind have been described as complicating both spontaneous and insulin-induced hypoglycemia. Stern, Dancey and McNaughton (15) noted a disturbance of cutaneous sensation in the extremities of 10 of 103 schizophrenic patients during the course of or following insulin-shock treatment, though in one case only were objective signs demonstrable. Five of these patients also suffered a disturbance of taste and smell Bückmann (5) reported 2 similar cases. In one a choreo-athetotic disturbance of gait was followed by a complaint of tingling in the finger tips and pains in the soles of the feet and calves. The other developed anesthesia and parasthesiae in the finger-tips together with cramps in the calves and slight right-sided cardiac enlargement. In both cases all the symptoms were transitory. The conclusion was that there was little doubt that these were beri-beri symptoms resulting from relative B1 avitaminosis in the presence of an abnormally high carbohydrate intake.

Kastan (11), after encountering another patient who complained of numbness of the fingers following insulin injection, examined the electrical reactions of 63 patients 2 to 4 hours after the termination of insulin coma. In a proportion of these a partial degenerative reaction with inversion of the type of muscle contraction was elicited. A similar type of investigation in the present case, however, showed no such abnormality.

Baker (1) quotes the case of a diabetic who in error received a gross overdosage of insulin resulting in a stupor of 10 days' duration, recovery from which was attended by a complaint of numbness in both hands and lower forearms unaccompanied by objective sensory disturbance. Apart from insulin overdosage, attention may be drawn to the frequent occurrence of parasthesiae in diabetes mellitus, burning sensations, numbness, tingling and a feeling of coldness in the extremities occurring in 70 per cent of Jordan's series (10). In a substantial number neuritic symptoms appeared while insulin was being given, in some cases shortly after it was first administered. Mental depression and emotional instability, though not disproportionate to the discomfort experienced, were also commonly observed.

In cases of spontaneous hypoglycemia the attack may be accompanied by parasthesiae of the tongue and lips (20), though this appears to be a transient phenomenon. Similarly when sensory symptoms in the extremities occur these usually subside, as in one of the series of patients reviewed by Wauchope (17) who developed tingling and numbness in both hands and arms, which persisted for 5 or 6 days after a severe attack. However in a patient of Willner and Weinstein (21), who started to complain of persistent numbness of both legs, feet and hands a few months after the onset of hypoglycemic attacks, these symptoms persisted until the removal of a pancreatic adenoma three years later, following which they subsided markedly despite the fact that abnormalities of the reflexes and certain objective sensory signs remained unchanged.

The pathologic basis of these symptoms is extremely difficult to explain. Polyneuritis may be a cause, though this explanation may be logically advanced only in those cases where the alteration in sensation is symmetrical, affects both upper and lower extremities, and is accompanied by objective sensory changes and signs of lower motor neurone involvement. Where, as in the present case, the symptoms are less widespread and unaccompanied by other signs such an explanation seems untenable. Nevertheless at the time the patient's

sensory symptoms first appeared she also complained of weakness of her legs and difficulty of gait, though this did not persist for any considerable time. Unfortunately there is no record of an objective examination of this complaint having been made at the time, but this history does suggest that initially a more widespread neurologic involvement was present. Furthermore if, as has been suggested, vitamin B1 deficiency is the basis of this type of disorder, the fact that the sensory symptoms may, as in the present case, be somewhat restricted, need not arouse undue controversy. Recent experience of nutritional disorders of the nervous system attendant on the semi-starvation of prisoners-of-war has shown that localised parasthesiaes and other neuritic symptoms are not uncommon (9).

Whether this view is correct or whether the neuronal damage can be ascribed to some toxic action of insulin either direct or due to interference with the oxidative mechanism of the nerve cells involved (7, 13), cannot at present be determined. Similarly there is no evidence in the present case, as has been suggested in some others (1, 2, 7), that vascular alterations played a part. The apparent irreversibility of the symptoms is no doubt related to the severe and prolonged nature of the hypoglycemic attacks, and emphasizes the need for early diagnosis in cases such as this, if complications are to be prevented. One other possible contributory factor may be considered. Although there was no clinical evidence of neurovascular compression at the shoulder girdle, it seems feasible that the slight abnormality of the 7th cervical vertebra that was present may have indicated a locus minoris resistentiae which might have determined the site if not the origin of the disorder.

The response of this particular patient to Chlorpromazine, although of interest, sheds no further light on the cause of the symptoms. It is clear that the drug acted by producing a state of psychic indifference to the discomfort, and that this beneficial effect can be expected to persist only so long as medication is continued. The failure of response to vitamin B1 merely indicates that if such a deficiency was the original basis of the complaint, the pathological changes produced are of an irreversible kind.

Summary

A patient suffering from persistent neuritic symptoms following severe spontaneous hypoglycemia is described.

The causes of this disorder are discussed in relation to observations made on other cases. Although the exact basis cannot be determined, it is thought that the symptoms may be the result of irreversible neuritic changes either due to sudden relative vitamin B1 deficiency associated with the abnormality of carbohydrate metabolism, or the result of some other direct or indirect action of insulin.

Comment is made on the alleviation of the patient's symptoms by Chlorpromazine.

BIBLIOGRAPHY

(1) Baker, A. B.: Cerebral damage in hypoglycemia. *Amer. J. Psych., 96:* 109, 1939.

(2) Black, K. O., Corbett, R. S., Hosford, J. P., and Aldren Turner, J. W.: Spontaneous Hyperinsulinism due to islet-cell adenoma. *Brit. M. J., 1:* 55, 1954.

(3) Brain, W. R.: *Diseases of the Nervous System,* 4th ed., 942. Oxford University Press, 1951.

(4) Brain, W. R., Wright, A. D., and Wilkinson, M.: Spontaneous compression of both median nerves in the carpal tunnel. *Lancet, 1:* 277, 1947.

(5) Bückman, I.: Beriberisymptoms bei der Hypoglykämietherapie der Schizophrenie. *Nervenarzt., 10:* 412, 1937.

(6) Crain, E. L., Jr., and Thorn, G. W.: Functioning pancreatic islet cell adenomas. *Medicine, 28:* 427, 1949.

(7) Ferraro, A., and Jervis, G. A.: Pathologic considerations on insulin treatment of schizophrenia. *Am. J. Psych., 96:* 103, 1939.

(8) Fraser, R., Maclay, W., and Mann, S. A.: Hyperinsulinism due to pancreatic islet adenoma. *Quart. J. Med., 7:* 115, 1938.

(9) Helweg-Larsen, P., Hoffmeyer, H., Kieler, J., Thaysen, E. H., Thaysen, J. H., Thydesen, P., and Wulff, M. H.: Famine disease in German concentration camps, complications and sequels. *Acta Psych. Neurol. Scand. Suppl. 83:* 215, 1952.

(10) Jordan, W. R.: Neuritic manifestations in diabetes mellitus. *Arch. Int. Med., 57:* 307, 1936.

(11) Kastan, M.: Electric corpse changes and reaction on the peripheral nerves in insulin-treated schizophrenic patients. *J. Nerv. Ment. Dis. 92:* 630, 1940.

(12) Lidz, T., Miller, J. M., Padget, P., and Stedem, A. F. A.: Muscular atrophy and pseudologia fantastica associated with islet cell adenoma of the pancreas. *Arch. Neurol. Psychiat., 62:* 304, 1949.

(13) Moersch, F. P., and Kernohan, J. W.: Hypoglycemia. Neurologic and neuropathologic studjes. *Arch. Neurol. & Psychiat., 39:* 242, 1938.

(14) Silfverskiöld, B. P.: Late peripheral paresis after hypoglycaemic attacks in two insulin patients. *Acta. Med. Scand., 125:* 502, 1946.

(15) Stern, K., Dancey, T. E., and McNaughton, F. L.: Sensory disturbances following insulin treatment of psychoses. *J. Nerv. Ment. Dis., 95:* 183, 1942.

(16) Walshe, F. M. R.: On "acroparasthesiae" and so-called "neuritis" of the hands and arms in women. *Brit. M. J., 2:* 596, 1945.

(17) Wauchope, G. M.: Hypoglycemia. Critical review. *Quart. J. Med., 26:* 117, 1933.

(18) Whipple, A. O.: Hyperinsulinism in relation to pancreatic tumors. *Surgery, 16:* 289, 1944.

(19) Wilder, J. Ein neues Hypophysäres Krankheitsbild. Die Hypophysäre Spontanhypoglykämie. *Deutsches Ztschr. Nervenh., 112:* 192 1930.

(20) Wilder, R. M., Allan, F. N., Power, M. H and Robertson, H. E.: Carcinoma of the islands of the pancreas. *J. A. M. A., 89:* 348 1927.

(21) Willner, V., and Weinstein, V. A.: Islet-cell adenoma of the pancreas with increased insulin excretion in the urine. *N. Y. State J. Med 50:* 1103, 1950.

UNIVERSITY DEPARTMENT OF PSYCHIATRY
THE ROYAL INFIRMARY
MANCHESTER 13, ENGLAND

SOCIETY PROCEEDINGS

THE NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY, AND THE NEW YORK NEUROLOGICAL SOCIETY

Joint Meeting, January 11, 1955

MORRIS B. BENDER, M.D., Chairman, Section on Neurology and Psychiatry, *presiding.*

RESULTS OF ANTERIOR CHOROIDAL ARTERY OCCLUSION IN PARKINSONISM. *Dr. Irving S. Cooper.*

It is the purpose of this presentation to present a follow-up study of the results of our personal experience with anterior choroidal artery occlusion in parkinsonism. To this end a motion picture was shown which demonstrates six parkinsonian patients preoperative, immediately postoperative, and at follow-up periods varying from 3 to 24 months. These films demonstrate immediate postoperative relief of tremor and rigidity, without motor loss, in the extremities contralateral to operation. Persistence of this alleviation of tremor and rigidity and improvement in activities of daily living are demonstrated in the follow-up motion pictures.

In addition, six patients are presented in person who were subjected to the anterior choroidal artery operation between 9 and 24 months previously. Each is demonstrated to have had alleviation of tremor and rigidity since operation. This documents the results recently described in a series of 50 consecutive operations of anterior choroidal artery occlusion.

Discussion

Dr. John Arthur MacLean: I would like to make a comment from the anatomic standpoint. I think we have all fallen into a bad fallacy in reading textbooks, anatomic, pathologic, and physiologic, that have been perpetuating anatomic inaccuracies.

Dr. Harry A. Kaplan: At Kings County Hospital a project has been carried out during the past 15 years concerning the therapy for Parkinson's disease. In connection with this, a study of the distribution of the arteries of the brain has been pursued. Naturally, we were quite interested in the procedure described by Dr. Cooper for relief of disabilities associated with Parkinson's disease.

On six patients occlusion of the anterior chorioidal artery was performed. Tremor and rigidity were substantially altered in only one of these. However, in this case, on emotional stress, the tremor would recur. In none of these patients were there bilateral effects, such as improvement in posture or gait. In two, tremor and rigidity ceased for 24 hours, then recurred with the same intensity as before operation.

A statement was made just now implying that the description of the distinction of the anterior chorioidal artery, as recorded in textbooks, was inaccurate. With this I disagree. An artery may be occluded surgically at a single point; however, the part distal to the site of occlusion may still function by virtue of its collaterals. The anterior chorioidal artery is no exception. It has many branches and receives collateral from at least four different arteries. The branches of this artery, which usually supply a small area of the mesial portion of the medial part of the globus pallidus, are not constant; this part of the brain may occasionally receive its blood supply from branches of the carotid itself or, in some instances, from the middle cerebral artery. One of our patients who was subjected to occlusion of the anterior chorioidal artery died six months later. At autopsy the surgical occlusion of the artery was found to be complete. The only lesion attributable to the occlusion was located in the most mesial inferior part of the globus pallidus and the immediate subthalamic area.

Dr. Cooper: I have little to add. It is true that these results have not been confirmed in all series, although many neurosurgeons have confirmed these results. In my own hands I have had some failures on a technical basis where I failed to completely occlude the artery. I think in other people's hands, when they have a failure, they should investigate that possibility. I do an angiogram before the case is operated and, if the operation has failed, I do a post-

operative angiogram. In some such cases we have been able to demonstrate that the artery is still open, and I think particularly in one's early experience of five or six cases, such as in Browder's experience, that would be a point to bear in mind. How often these good results can be reproduced I do not know, although the percentage is high in our own series. We have shown 7 or 8 patients in the movies who are free of tremor or rigidity, and in addition the 5 patients who were personally demonstrated tonight conclusively show excellent results. If time had permitted we would have had many more such results here. My purpose was merely to present in person these unequivocal facts and results. We must wait for a longer time before concluding how often the results can be reproduced, and how long the duration will be.

A Psychological Study of Visual Auras in Patients with Brain Damage and Seizures. *Dr. Thomas C. Guthrie* (by invitation) and *Dr. Aaron T. Beck* (by invitation).

The five patients included in this investigation demonstrated evidence of gross structural disease of the temporal or occipital cortex. Associated with the brain damage were grand mal seizures preceded by a visual aura. Each patient presented with a history of head injury and had neurological deficits on examination and an abnormal electroencephalogram. In studying these patients, psychiatric interviews, psychological tests, hypnosis, and free association amplified the neurological evaluation. It has been possible to reproduce the visual aura under hypnosis, without the seizure. When hypnotized, these patients revealed much fantasy material related to their visual hallucinations.

From these experimental data we have concluded that the visual aura, in brain-damaged patients, is a manifestation not only of the patient's lesion, but is also related to the personality of the individual. The visual aura is an expression of the individual similar to a dream, and employes psychological mechanisms such as symbolization and distortion. The visual aura and subsequently the seizure occur at a time when certain threatening or disturbing life situations arise. The visual aura then is not formed exclusively by activity in a discrete area of cerebral cortex but is a complex phenomenon resulting from the total integrative activity of the brain.

Discussion

Dr. Donald J. Simons: I think it would be interesting to know whether the treatment of the combat neurosis in the first patient made any change in the nature of the patient's seizures.

Dr. Stanley Lesse: I think it is interesting to note that these visual aura occurred in brain-damaged patients; in nonorganically ill patients (schizophrenics) we have stimulated the temporal lobes in approximately 15 cases under local anesthesia; and we have not been able to reproduce any of the visual or hallucinatory material.

Dr. Guthrie: In answer to Dr. Simons' question I would like to state that with psychotherapy and anti-convulsants the patients all improved. They had fewer seizures, but I would not be able to make any statement as to which agent was responsible for the improvement.

The Use of Large Doses of Cortisone in Schizophrenia. *Dr. Phillip Polatin, Dr. Stanley Lesse* (by invitation) and *Dr. Meyer M. Harris* (deceased).

Nine non-deteriorated schizophrenic female patients were given 500 mg. of cortisone daily for from 4 to 10 weeks. These patients were studied intensively from a psychiatric, physiologic and laboratory point of view. Three patients manifested slight but transient psychiatric improvement. Five were definitely made worse and one seemed unaffected. Many presented an increase in psychomotor activity and a marked increase in anxiety. No significant long-standing beneficial therapeutic results were obtained using cortisone alone. Cortisone did not appear to work synergistically with electroshock therapy Six patients revealed clinically demonstrable physical changes with the massive doses of cortisone, although only two showed a modified Cushing's syndrome. It was surprising to record that no significant alterations in electrolytes or other blood chemistry studies were seen. A significant eosinophil drop occurred in six patients. Two other patients manifested a definite lowering of the basal metabolic levels. The most startling result of these studies was the lack of more pronounced evidence of hyperadrenalism in more of these patients due to the massive doses of cortisone. To explain this it is postulated that huge amounts of cortisone may possibly produce an adverse reaction in the anterior pituitary which in turn may inhibit normal pituitary-adrenal relationship, or else there is the possibility that the endocrinological organization of schizophrenic patients is so constituted as to be uniquely refractory to massive doses of cortisone.

Discussion

Dr. Paul Goolker: We are all grateful for the facilities provided by the Psychiatric Insti-

tute for the testing and assaying of new drugs and methods of therapy, and for the way Dr. Polatin and his collaborators have utilized these facilities for our enlightenment. The study of the use of drugs, in addition to contributing to therapy, provides a means for the further understanding of psychic function, and this is just as important as the therapeutic measures that are applied.

Dr. Stanley Lesse: I would like to thank Dr. Goolker for his discussion of our paper. There are many valid criticisms and questions offered by Dr. Goolker: 1) we had too few cases, but we had only a certain amount of drug to use. 2) The reason why we chose 500 mg. was not purely an abstract choice. We had had a verbal communication from Cohen in Cleveland, stating he had been using this dosage, and he felt that with large doses he had gotten his best results. That was the reason we used this dose.

GROUP PSYCHOTHERAPY IN PATIENTS WITH PEPTIC ULCER. *Dr. Aaron Stein.*

In an effort to deal with the underlying emotional tensions which, according to present views, are thought to be similar in most patients with peptic ulcer and which are one of the factors causing their symptoms to persist, a group of patients with chronic peptic ulcer was treated for a period of 18 months with a psychoanalytically oriented type of group psychotherapy. All of the patients were found to have severe underlying psychiatric disease with rigid constricted personalities and poor object relationships. In 14 of the 15 patients the onset of ulcer symptoms seemed related to the loss or threatened loss of the mother or mothersurrogate.

Because of the supportive and tension-lessening factors operative in the group psychotherapy, all of these rigid constricted patients participated actively in the treatment. After working through their guilt over being ill, they were able to express, first, resentment over not being cared for, and then to verbalize their dependent needs. Slowly, the rigid intolerant group attitudes changed to more tolerant and flexible ones so that the patients could begin to develop more realistic relationships and more effective methods of satisfying their needs.

Of the 15 patients in the group, 12 continued in the group treatment. Three of these 12 were considered unimproved, the remaining 9 showed varying degrees of improvement, both psychiatrically and in their ulcer symptoms. The degree of psychiatric improvement in general tended to parallel the degree of improvement in the ulcer symptoms.

It was concluded that group psychotherapy appears to have definite usefulness in aiding patients in whom chronic peptic ulcer is associated with a regressed, rigid type of personality.

Discussion

Dr. Jacob A. Arlow: Twelve to 15 patients observed for a period of almost two years, with several years of ensuing follow-up is a considerable psychotherapeutic experience. I doubt if the original studies which formed the basis for the psychodynamic formulations of peptic ulcer had a larger group of patients.

Concerning the selection of the patient the following must be noted: there were two sets of criteria used—one furnished by the physicians, namely, the presence of peptic ulcer, and other furnished by the patients, namely, *their* willingness to participate in a program of group psychotherapy. It would seem to me that at this point a very significant weighting was introduced into the sample.

The very high incidence of severe psychopathology (100 per cent) in this group may be attributed to this unwitting deviation of a selection of patients in the direction of the emotionally ill. It is most doubtful that a random selection of ulcer patients in general medical practice would reveal them to be uniformly rigid, immature, dependent emotional cripples. In addition to the therapist's experience and skill (Dr. Stein has been pioneering in this field for many years) the receptiveness of the patients so selected is another factor which contributed to the truly impressive therapeutic results reported in this paper.

NEW YORK NEUROLOGICAL SOCIETY AND THE NEW YORK ACADEMY OF MEDICINE, SECTION ON NEUROLOGY AND PSYCHIATRY

Joint Meeting, February 8, 1955

E. JEFFERSON BROWDER, M.D., President, New York Neurological Society, *presiding.*

THE VALUE OF RADIATION THERAPY IN THE MANAGEMENT OF GLIOMA OF THE OPTIC NERVES AND CHIASM. *Dr. Juan M. Taveres* (by invitation), *Dr. Ernest H. Wood* (by invitation), and *Dr. Lester A. Mount.*

Primary tumors of the optic nerves and chiasm are encountered infrequently, with the result that the effect of various forms of treatment is difficult to evaluate. In the past, emphasis has been placed upon surgical treatment, and there is very little indication from the literature how important radiation therapy may be in the management of such neoplasms.

Preliminary observations in the results of treatment of 34 patients with optic nerve glioma at the Neurological Institute of New York and the Institute of Ophthalmology indicate that roentgen therapy is of foremost importance in the control of optic nerve glioma, particularly when combined with conservative surgical treatment. Radical surgery usually results in permanent destruction of the optic pathways or in an increase in the visual deficit caused by the tumor. Radiation therapy, in some instances, has resulted in a restoration of vision, while on other occasions advancing visual impairment caused by the tumor has been arrested. In some of the patients with exophthalmos, the globe has receded completely and permanently. Large tumor doses often are not necessary to produce beneficial effects. The best results have been observed when radiation therapy has been used as the primary method of treatment and surgery has been reserved for those cases in whom there is obstruction to the flow of cerebrospinal fluid or when an existing exophthalmos fails to respond to radiation therapy.

Discussion

Dr. Lester A. Mount: There are a few points which I should like to emphasize. First, surgical therapy is indicated in those patients in whom a positive diagnosis cannot be made. This includes the cases in whom the tumor is entirely intracranial, having no enlargement of the optic foramen and no exophthalmos. In these cases exploration is indicated to verify the tumor before starting x-ray therapy, but if the tumor is found to be entirely within the optic chiasm and the optic nerves no biopsy should be taken. Of one patient, upon whom I operated eight years ago, the only present visual impairment I believe to be the result of the biopsy which I took from his chiasm.

It includes also the cases in whom the tumor is entirely within the orbit, having exophthalmos and no enlargement of the optic foramen. Surgical treatment in these patients is an intraorbital exploration before starting x-ray therapy, and if the tumor is found to be within the optic nerve no biopsy should be done.

Operative therapy is necessary in the patients in whom there is an obstructive hydrocephalus before starting x-ray therapy. A by-passing procedure is the treatment of choice, as the direct attack on the tumor in an effort to relieve the obstruction usually proves fatal.

A further possible indication for surgery is the case in whom the tumor is entirely within the optic nerve. However, such a course would result in the loss of vision in one eye and it is doubtful that the tumor would grow beyond the point where it could be removed completely in the 28 days necessary to administer 4000 r. of x-ray therapy and two weeks thereafter. The patient could then be re-evaluated and if he has not improved, operative therapy would then be imperative.

Dr. George H. Hyslop: As some of you may know, Russell Meyers of the University of Iowa has an interest in semantics. At Memorial Hospital some of these tumors which you call optic nerve gliomas are not known are retinoblastomas. When we find it is a retinoblastoma it is too late for x-ray, so far as we know now. One of the problems of the so-called optic chiasm glioma, and you can diagnose that on the basis of a proper history, excluding a retinoblastoma and excluding x-ray abnormality, is whether it is radiosensitive.

DIURNAL RHYTHM IN EPILEPSY. *Dr. Max Levin.*

Epileptic fits do not occur at a uniform rate throughout the day and night. On the contrary, they show a preference for certain hours; these are the late hours of sleep and the early hours of the morning after waking up. The reason for

this would seem to lie in the interplay of excitation and inhibition. Excitation and inhibition are constantly counteracting each other, as though in an unceasing struggle. Of course, sooner or later inhibition is "victorious," for sleep (an inhibitory state) is inevitable. At the end of the day excitation has, so to speak, exhausted itself and inhibition takes over. But after a few hours the position is reversed; during sleep the potential for excitation builds up till it reaches a peak when we wake up, ready for the new day. The cycle is like a ferris wheel that spins once a day. At the start of the day the potential for excitation is at a peak; at the end of the day it is all the way down. The epileptic fit is an excitatory phenomenon, and is therefore more likely to occur in those hours when the potential for excitation is highest.

Discussion

Dr. Leo L. Orenstein: The author by presenting his material develops his theory of "excitation and inhibition." Dr. Levin even makes use of his understanding of football to illustrate his clinical observations. I like football very much, and if I felt it could enrich my insight into clinical problems, I would attend more games.

There is one disturbing clinical problem, namely the patient suffering from depression, who frequently is not able to sleep. This patient at the same time shows ample evidence of inhibition of motoric and mental functioning. If in terms of the theory presented tonight excitation leads to activity, and fits, and inhibition to a state of sleep, as in narcolepsy, then I wonder if the author would care to comment on the paradoxical findings in the patient with depression.

Dr. Levin: I won't try to answer the question raised by Dr. Orenstein. The problems of depression are infinitely more complex than those of narcolepsy and epilepsy. These last two disorders are relatively simple, and their manifestations can be accounted for by the principles of excitation and inhibition. The problems of depression are of a much higher order of complexity.

HOSPITAL TREATMENT OF ANOREXIA NERVOSA. *Dr. James H. Wall.*

This study is concerned with practical experience in the hospital treatment of ten patients suffering from anorexia nervosa. The pertinent literature is reviewed. The family background, personality development and symptomatology of the patients are presented. Little more has been added to our understanding of the syndrome since it was described by Sir William Gull in the last century but there has been an increase in the study of the condition, paralleling the revival of the interest in psychosomatic disorders. The ten patients, girls and women, were between the ages of 16 and 38. They moved from one level of adjustment to another with great difficulty and feared growing up and assuming the responsibilities and privileges of adulthood. They repudiated life and sexuality, and the themes of denial and renunciation became most prominent at the time of puberty. As a group they were restless but shallow, withdrawn and infantile. This group resembled the sweeping total reaction of simple schizophrenia. Treatment includes tube-feeding, ambulatory insulin, electric shock therapy, psychotherapy and socialization through the various program therapies. This group made rather limited adjustments after prolonged periods of treatment. Seven of the ten have done well, but they continue under-weight and all find it difficult to achieve a weight over 100 pounds.

Discussion

Dr. M. Ralph Kaufman: We, at the hospital, have been interested in the problem of anorexia nervosa for a number of reasons. One is that the syndrome has remained essentially unaltered since its description by Sir William Gull in 1873. There are now about 400-plus papers relating to anorexia nervosa in the literature, and essentially the syndrome as originally described remains the same. There have been many different attempts to explain the syndrome; in recent years there has been an emphasis on certain psychodynamic aspects of the clinical syndrome. All workers have emphasized that in the psychiatric picture one can make, or does make, diagnoses as varying as hysteria, compulsive and obsessive neuroses, schizophrenia, manic-depressive, and so on, but nevertheless the central elements of anorexia nervosa are the same. It seems as though what one deals with is a syndrome which has a certain specific pattern in a matrix which can be anything from schizophrenia to hysteria. The difficulty in making a diagnosis in any given case as from one hospital to another is rather well known. It is not because one hospital knows how to make the diagnosis and the other does not, but because we deal with a series of defensive maneuvers that are in a dynamic flux and may change at any point in time.

It does seem a little peculiar to me that none of the patients of Dr. Wall should have had any oral impregnation fantasies, since all 24 of the patients that we have had had fantasies of oral impregnation that were so clearcut that we rather took it for granted.

Dr. Oskar Diethelm: In the 45 cases which were studied at the Payne Whitney Psychiatric Clinic there have been various groups, including schizophrenic reaction, hysterical reactions, characterized by phobic "anorexia," and compulsive symptoms. It was of interest to me, in going over our material, to note that the least desirable results were obtained in patients who were under 17 years of age. They comprised about 20 per cent of our patients. With two exceptions they all had a compulsive type of anorexia nervosa, but seemed to belong, with those two exceptions, in a schizophrenic entity. I think they were all sent to boarding school or special schools, but with a rather disappointing result. In the cases which Dr. Wall discussed, there was one of our former patients included. Good results were obtained in the hysterical reaction of the phobic type of anorexia nervosa in our patients over 20 years of age. In the follow-up study, some including 15 years' duration, the results were good. In these cases long-term psychotherapy was carried out; one patient stayed 14 months in the Clinic for intensive psychotherapy. A certain group included patients who had a compulsive type of food refusal. The outcome was rather interesting. One or two may have had paranoid schizophrenia; others developed rather simple deterioration, some in the hospital, some outside; one received lobotomy and, as was anticipated, without much benefit.

THE NEWEST TRENDS IN FORENSIC PSYCHIATRY. *Dr. Gregory Zilboorg.*

The influence of psychiatry has made itself felt in the handling of medico-legal problems of a psychiatric nature. This influence is reflected in such matters as "lie detectors" and various drugs which came into medico-legal practice via the so called narco-analysis or narco-synthesis The crucial question in such matters is whether or no the physiological and/or pharmacological tests are of really evidential nature. No one denies the psychological importance of these measures, but it is questionable whether the amytal or pentothal tests are in any way admissible from the purely legal or moral point of view. After all, a person under the influence of a drug is an *altered* person, and therefore is no the same as the one who committed the crime or who happens to be under consideration. The Yale medico-legal unit does interesting work on this subject, but its conclusions are far from definite or convincing as yet.

The second aspect of psychiatric influence on forensic psychiatry is reflected in the recent decision of the Monte Durham case by the U. S. Court of Appeals of the District of Columbia. This decision is identified with Judge Bazelon who wrote the opinion, with Judges Washington and Edgerton concurring.

The major feature of this decision is that it does away with the right-and-wrong test, with the irresistable-impulse theory, and in general, with purely legal definition of so-called insanity. Instead it establishes the sound principle of psychiatric criteria and asks the psychiatrist to tell the court and the jury whether the given crime is a direct result of the given mental illness of the given defendant. This is a tremendous advance, but it has its pitfalls in that it throws even a greater burden on psychiatry. However, it has the enormous advantage over the old McNaghten rule in that it eliminates the double standard of insanity—legal and medical—and also frees the psychiatrist from passing on the question of criminal responsibility which is not a psychiatric question.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Meeting of May 20, 1954

DR. WALTER WEGNER, *President, presiding.*

ANATOMICAL OBSERVATIONS OF FRONTAL LEUKOTOMIES WITH SPECIAL REFERENCE TO THE ORGANIZATION OF THALAMIC AND CORTICAL PROJECTIONS TO THE FRONTAL LOBE. *Paul I. Yakovlev, M.D.*

A study of the cerebra from patients with a sufficiently long postoperative survival following frontal leukotomy revealed that the surgical lesions restricted to the frontal pole and the anterior convexity of the frontal lobe do not result in a significant degeneration of the mediodorsal nuclei of the thalami; in such specimens there is also no degeneration of the fronto-pontine tracts. On the other hand, the cerebra in which the opercular and the orbitomesial paralimbic cortical areas bordering on the Island of Reil and the limbic lobe were damaged showed, consistently, degeneration of the mediodorsal

nuclei and of the fronto-pontine tracts. On this and other evidence it is proposed that the areas of cortical termination of the thalamo-frontal projections from the mediodorsal nuclei and the areas of cortical origin of the fronto-pontine tracts coincide and that in man the fronto-polar and anterodorsal convexity of the frontal lobes is relatively "a-thalamic" and "a-pontine." The thalamic nuclei appear to project upon the cerebral cortex in an orderly spatial sequence following the chronogenic sequence of the evolution of the cortical areas of their projection: the anterior and the medioventral groups of nuclei project mainly to the limbic cortex and to the Island of Reil; the mediodorsal nucleus projects upon the opercular and paralimbic areas bordering on the limbic lobe and Island of Reil; and the posterolateral groups of nuclei project chiefly to the cerebral lobes situated behind the plane of Rolando fissure. The anterior convexity of the frontal lobe represents thus the so-called frontal association area which has, even in monkeys, barely emerged from the thalamus-bound limbic lobe, but which in man has achieved a nearly complete freedom from the thalamic bonds and is surrounded laterally, basally, medially and posteriorly by the areas of cortical "conjugation" of the relatively abundant thalamocortical projection fibers with the long efferent fiber tracts from the frontal lobe to the striatum, the brain stem and the spinal cord. The frontal lobe, comprising the pre-Rolandic half of the hemisphere, represents thus the origin of the great common path open to all modalities of human experience, the cerebro-peduncular path. The post-Rolandic half of the hemisphere represents on the other hand the central-most end of all the private paths. The inexorable privacy of any personal experience, however intimate and non-sharable, can be breached and the experience made public only if that experience is somehow translated into some sort of movement—a motor act; only thus, through movement, a private experience becomes public, i.e., common and therefore sharable experience. Following the logic of inference from anatomical evidence, the frontal association area appears to be the instrument for that translation of the intimately private personal experience into the irrevocably final and forever irretrievable experience of public action in and through which the private non-sharable experience becomes, like a cat let out of the bag, a common experience of change and motion sharable by all. Even in the highest of primates the privacy of personal experience, if we assume that there is any, is all in the overt, immediately sharable action. Not so in man. Emancipated at least in his frontal association areas from the thalamic tutelage over his brain, he can be master of even the wildest cats he may hold in his private bag. This specifically human area of the brain may well be regarded as the cerebral correlate of socially adapted action symbolized in abstract thought, expressed in a meaningful language and materialized in relevant manufacture, i.e., in the products of thoughtful action.

Discussion

Dr. Hannibal Hamlin: I do not think that the neurosurgeons involved with this material can feel proud of the first slide which showed so much variation in the direction of the lobotomy incision. It makes us realize that some of the variation in the findings of Dr. Yakovlev were probably caused by the hand of the surgeon. But the basic facts derived from the long-term study of these specimens is that post-leukotomy degeneration in the thalamus is not as important in relation to frontal cortex as previously supposed. The more we review these excellent studies of Dr. Yakovlev, the more conscientious we should become in performing lobotomies. I supplied a few of these specimens from the days of early experience and I am grateful to learn of their final and valuable disposition.

Dr. Stanley Cobb: Dr. Yakovlev's papers are always interesting, not only because he knows the anatomy and shows it with such unusual art, but because there is a lot of real thinking behind them. In this case he made me think about a recent book I have been reading, by Penfield and Jasper. In that, you will remember that there was a lot of weight put on the centrencephalic system. You have showed us this evening that there is an area in the convexity in parts of areas 9 and 10 and perhaps 11 where there isn't any immediate relationship in this cortex with the diencephalon. Therefore your work could be used as an argument that this idea of the centrencephalic system as the highest level of integration is probably not so.

Dr. Paul B. Jossmann: The clinician's interest in the working mechanism of lobotomy is highly stimulated by this paper. A revision of the hypothesis seems indicated by the fact that "In man the anterolateral convexity of the frontal lobe appears to be relatively 'a-thalamic' and 'a-pontine'." These remarkable findings seem to

indicate that the working mechanism of lobotomy does not consist alone of severing the frontal-pontine and the thalamofrontal projections. Also, Dr. Yakovlev's findings represent a challenge to explaining the working mechanism of other types of psychosurgery, like the topectomies.

Dr. James C. White: One can easily avoid inaccurate placement of the lobotomy incisions, as shown in Dr. Yakovlev's specimens, by following Grantham and Spurling's techniques. Through superior parasagittal trephine openings a small amount of air is injected into the anterior portion of each lateral ventricle. The ventricular needle is then inserted so that it presses just in front of the tip of the ventricle and rests on the bony floor of the anterior fossa just rostral to the sphenoid ridge. After checking its position by x-ray one can destroy the desired area of frontal white matter by suction or electrocoagulation.

Dr. Yakovlev: The ideas promulgated by the Montreal School of Neurology, on the notion of the centrencephalic system as the highest level of function and the central mechanism of consciousness, are stimulating and instructive. The difficulty with the notion is in the value judgement which it irresistibly compels. I am very much tempted to try to place the problem of the organization of functions not in terms of "hierarchies" of functions "from below up" but in terms of chronogeny of their evolution "from within out." In this scheme of thought the centrencephalic system of Penfield, as I understand it, would correspond then to the headmost part of the diffuse system of short neurons assembled at *random* nearest the primordial hollows of the neuraxis where this system is anatomically seen with pristine clarity although in fact it permeates the entire neuraxis. This system is concerned with the maintenance of the status quo within the body against those forces of change which are universal for all living systems and which have been in operation ever since there was life on earth. The changes which these forces constantly tend to produce in the living systems are long-term, repetitive changes *predictable for all time* as are predictable the slow, unalterable and repetitive succession of days and nights, of seasons and of tides. Within and from this *innermost,* essentially reticulate system of primordial visceration consisting of short and random relays there developed in the vertebrates the *intermediate* system of no longer random but space committed long relays disposed *in series.* This discretely nucleated system develops in the mantle zone of the neuraxis and in the main therefore is external to the innermost system. This intermediate system is concerned with the maintenance of gravitational and thermodynamic status quo within the organism against those forces of change in these equilibria which, while predictable for all time, are fast, short term changes and which therefore allow no choice in the means of adaptation to them except those means which are inherent in the organization of the species—in vertebrates the skeletal musculature. This system regulates the postural adjustments of parts of the body wall with its appendages to the body itself in stance, locomotion, body attitudes, gesture, mimicry, vocalization and all movements of expression of internal states—*the emotions.* The headmost part of this system of emotional expression is the limbic lobe with the thalamus and the striatum interposed between. And in the marginal zone there develops in mammals the *outermost* system of long relays connected *in parallel* to ever larger numbers of modal points in the intermediate and the innermost systems. The headmost parts of this outermost system in mammals is the supralimbic cortex from which in primates and man emerge the associative areas of the frontal and temporoparietal lobes. This outermost system of neurons connected in parallel to the rest of the neuraxis is no longer concerned with the maintenance of merely status quo but precisely with the elaboration of the *new* modes of adaptation through choice and combination of whatever available means of adaptation to meet the challenge of ever new, constantly emergent and *altogether unpredictable* events of the everchanging world about the organism. Which of these three systems is lowest or highest in hierarchy is rather irrelevant. One may say the outermost system is more "sublime," but then the innermost system is more "vital." All three have their headmost components which are "highest" only in the sense of our human "verticality," but they are "foremost" in chronogeny of their evolution, and here the frontal association area is certainly the "foremostest." It does not complete its development until midlife of man—the age of discernment when his innermost system of visceration (the "centrencephalic system") and his intermediate system of emotion may have begun already to regress and atrophy.

PSYCHIATRY IN A GENERAL HOSPITAL. *William W. Sargent, M.B., M.R.C.P.,* Professor of Psychiatry, University of London, London, Eng.

No abstract of this paper is available.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Meeting of November 18, 1954

DR. WALTER WEGNER, *President, presiding.*

AUDIO-MOTOR MECHANISMS IN EXTRA-TEMPORAL CORTEX. *J. K. Merlis, M.D.,* and *C. Lombroso, M.D.*

Detailed mapping of the cortical electrical responses evoked by acoustic stimulation in the cat reveals an area of consistent response in the anterior suprasylvian region. This area is topographically distinct from the three other auditory areas previously described, and acoustic potentials are evoked in it after ablation of the other responsive regions.

The suprasylvian area of each hemisphere is excited by the contra lateral cochlea, whereas the ipsilateral cochlea appears to exert an inhibitory influence. The evoked potentials are insensitive to strychnine until this inhibitory influence is removed by destruction of the ipsilateral cochlea, whereupon they show a marked strychnine augmentation.

Observations of electrical stimulation of this region reveal that this is an area of low threshold for contralateral pinna movements, whereas stimulation of the primary auditory area in the ectosylvian gyrus evokes bilateral pinna movements. In addition to the contralateral pinna response, suprasylvian stimulation frequency evokes conjugate deviation of the eyes to the opposite side.

Synthesis of the data obtained with respect to the afferent and efferent mechanisms indicates that this suprasylvian area may be concerned with focusing attention to specific regions of the outside world, in contrast with the ectosylvian area, concerned with generalized alerting.

Discussion

Dr. Walter Wegner: In the middle 1800's, there was a great deal of discussion about how the brain worked. The original investigators maintained that the brain worked as a whole. Beginning around 1860 investigators began to realize that different parts of the brain had specialized functions. After those investigations were confirmed and accepted, clinical localization of nervous disturbances could be made. It would seem that we are now going in the reverse direction, that we are getting various functions mixed together again in a given area instead of being geographically isolated. Maybe the functional localizations we talked about will have to be looked at again. I have found this paper very interesting, and a couple of questions occurred to me as Dr. Merlis gave it. One, regarding the cytoarchitecture in these areas: is there a close resemblance in the ectosylvian as compared with the suprasylvian area? Second, is he acquainted with any human case material to which this work applies? We commonly believe that destruction of the auditory cortex in the temporal lobe on one side does not alter hearing significantly. Is there any case material in the literature which describes bilateral temporal lobe lesions? What happens to the hearing in these patients?

Dr. Warren McCulloch: There is a point which should be raised here, particularly with regard to bilateral lesions, not in the auditory cortex, but of the area anterior to it. Professor Galvardin of Lyons became interested in the same problem his father had worked on, i.e., auditory hallucinations which begin as arteriosclerosis in the latter years of life. This is accompanied by overt movements in the muscles in the tongue, pharynx and larynx. He selected one case for surgery. The man suffered from eternal chickens that informed him he was a good man, etc. They had become such a nuisance that he had himself incarcerated. Dr. Galvardin had removed one side of the face cortex with transient relief. This cortex lies behind the lower end of the Rolandic fissure and down into the border of the Sylvian fissure. Following the removal of the same cortex on the opposite side all movements in the pharynx and larynx ceased. He no longer heard the chickens although they "meant" what they always had meant.

Dr. Merlis: I am not an expert in architectonics but I can cite Rose's data which indicates that there is a change in crossing the sulcus. Ectosylvian gyrus is distinctly different in its cellular structure from suprasylvian. As for the influence of temporal lobe lesions on hearing in man, in the experimental primate, the monkey, removal of the superior temporal gyrus, and the posterior insula bilaterally, does not abolish appreciation of sound. It seems to be reasonable that this might also be true in man.

VISUO-MOTOR FUNCTION IN THE CEREBRAL CORTEX. *D. Denny-Brown, M.D.* and *R. A. Chambers, M.D.* (Boston)

Disturbances of visual perception (agnosia) result from damage to parastriate areas of the occipital cortex, in which areas concerned in visuo-spatial aspects of perception and of object recognition by vision have been postulated. Studies of monkeys after complete removal of area 17 by suction, leaving areas 18 and 19 intact, showed that prehensile reactions could be stimulated by moving objects, and placing reactions to surfaces and edges could still be elicited. Optic righting in space was also intact. Bright illumination inhibited these reactions. Visuo-mimetic behavior and visual recognition of still objects was absent. The lateral geniculate bodies were completely degenerated. Removal of areas 18 and 19, leaving area 17 intact, impaired spatial judgment, and led to confusion by moving objects, though placing and prehensile reactions remained intact. The posterolateral nuclei of the pulvinar were degenerated.

Removal of the lower part of areas 18 and 19 and the temporal lobes, as well as area 17, left optic prehensile and placing reactions, and optic righting intact, and abolished fear from visual stimuli. Linear visual contrast was the adequate stimulus for spatial and prehensile responses. Removal of the upper parts of areas 18 and 19 and the parietal lobes, as well as area 17, abolished visual prehension, placing and righting but left strong fear and flight reactions, especially to brightly illuminated surfaces. Dark contours then offered attraction.

These experiments indicate that there is a double visuo-motor mechanism in the cerebral cortex, one relating to the detailed perception of still objects, the other to moving objects. In each there is a horizontal organization into an upper segment which, with the posterior parietal lobe, is related to spatial reactions, and a lower relating to tropisms to objects. Each is independent of the other. The lower segments of areas 18 and 19 and the temporal lobe serve a powerful negative (avoiding) response to illuminated objects. There was no evidence of a separate area for spatial or object recognition, though the mechanism is such that separate disorders of these can be produced by small bilateral lesions.

Dr. Paul B. Jossmann: Observations on humans with visual agnosie confirm Dr. Denny-Brown's experimental work. Recognition and identification is facilitated when objects are in motion. Experiments undertaken with normal individuals show that on exposure of cinematographic films moving objects are recognized better than non-moving objects. I should like to ask whether the experiments have indicated a specific relation of gnostic functions to areas 18 and 19 Brodmann.

Dr. Robert Schwab: Does Dr. Denny-Brown have any idea which system is involved in stereo-vision, which these monkeys evidently have?

Dr. Walter Wegner: The lesions Dr. Denny-Brown has demonstrated in these monkeys were bilateral lesions. He has also been interested in the visual phenomena resulting from lesions in these areas in human beings and many of such lesions are unilateral. I wonder if he would comment on his observations regarding the visual phenomena in unilateral lesions in these patients. Some years ago a case of Dr. Dandy's, I believe, was reported in which he approached a third ventricle tumor through the posterior part of the corpus callosum and noted afterward that his patient was unable to recognize objects which appeared in the left visual field. If a friend approached from the left side, he would not recognize him but would recognize him readily when he got into his right field of vision. The surgeon presumed that this defect was due to cutting some fibers in the corpus callosum. It might equally well have been due to damage by retraction or contusion of the right hemisphere. In any event, it would suggest an interpretative function in the right hemisphere which we ordinarily believe resides in the left hemisphere.

Dr. Denny-Brown: To take Dr. Wegner's question first, I am sure these disturbances can be shown by unilateral lesions, but they are difficult to test in a monkey because he does not keep his eyes fixed. We have in the ward at present a patient with complete hemianopia for object recognition, as a result of occipital meningioma, but who can pick up movement in any part of the affected field of a quite small object. She could not tell you it was square or round, but only that there is something moving. It is difficult to get exact anatomical evidence. I would suspect area 17 had been damaged on one side in Dandy's patient.

As to difficulty to identify persons, most writers do not make movement an important factor. But in Dr. Jossmann's patient and in one described by Holmes and in others I have seen but not studied in detail, movement is obviously very important. I would think that one trouble in investigating agnosia is in knowing where to begin. It is like being presented with a mass of detailed things and trying to sort them out. The criteria chosen for current classifica-

tions may not be of importance in the nervous mechanism. The present work aims to define the most elementary physiological factors in visual recognition, and relate them to specific parts of the mechanism. The finding of two distinct vortical mechanisms, one related to the geniculo-striate projection, the other to pulvinar-parastriate projection, gives a new approach.

I do not know about stero vision in monkeys. It is difficult to judge depth perception in an animal. Some clearcut clinical cases would be necessary to decide that question. At present we do not even know why it is that the object has to move to be seen. Movement of the head, so as to produce apparent movement of the object, is not effective.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Meeting of December 16, 1954

DR. WALTER WEGNER, *President, presiding.*

MALFORMATIONS OF THE NERVOUS SYSTEM. *Paul I. Yakovlev, M.D.*

A series of specimens showing circumscribed defects in the cerebral mantle are shown and the evidence presented that these defects in structure of the brain are laid down not later than the end of the second month of gestation. The common characteristic of these defects is their tendency to occur in the zones of the primary fissures (sylvian, rolandic, medial orbital), and especially in the midline in the zone of the telencephalon medium and roof plate of the prosencephalon.

The available material illustrates all degrees of transition—a gradient of severity—of the malformation of the hemisphere from a simple agenesis of the corpus callosum, consisting of a failure of the commissural fibers to cross the midline but without any other abnormality in the structure of the cerebral hemisphere to a more or less extensive symmetrical defect in the development of the cortical plate amounting in some instances to a complete agenesis of the cerebral mantle.

There exists a tendency to confuse the two distinct issues, namely the issues of the *causal* and of the *formal* origins of malformations, and to search for a unique causal mechanism for all such defects. The so-called "vascular theory," claiming the failure of development of blood vessels supplying various parts of the cerebral mantle to be the cause, is most appealing to many. Yet, a careful study of the morphology and architecture of the forebrain in these malformations permits one to assert that blood vessels fail to develop because of the primary failure of organogenesis of the brain.

Whatever may be the causal origin of these defects, the key to the formal origin of these malformations is most likely to be found in the interference with the normal processes of organogenic induction upon which depends the regional differentiation of the brain.

Discussion

Dr. Walter Wegner: It is easy to see how Dr. Yakovlev's study of neuroanatomy has led to an interest in the study of malformations. Dr. Yakovlev has told us that these malformations depend upon influences which occur early in the life of the fetus. No doubt there are other malformations or other deformities which occur later due to noxious influences. I have wondered about the vulnerability of the brain at different periods. It is more vulnerable in the early periods than later? Is there evidence that, if the fetus is exposed to radiation during the first few weeks of gestation, it is more vulnerable than later on? Or, if the mother contracts measles, is the fetus more vulnerable at one period than another?

Dr. William H. Sweet: If I understand Dr. Samuel Hicks correctly, he states that different noxa at the same stage of embryonic development tend to produce different types of malformation or defect. Conversely, the same type of malformation may be produced at different stages in embryonic development by appropriate variation and dose of the deleterious agent. (I am referring to his paper "Injury of the Central Nervous System Incurred during Fetal Life" presented at the December, 1954 meeting of the Association for Research in Nervous and Mental Disease.) Is there any difference between his point of view and yours, Dr. Yakovlev?

Dr. Clemens Benda: Dr. Yakovlev's masterful contribution is a great stimulus to anyone engaged in this field. I would like to say a few words about the practical importance of those congenital malformations. Many physicians think that this is a rather limited field of special interest. Studies on our autopsy material of now

more than 300 cases indicate that about 30 per cent of children and adults suffering from severe mental defects have some type of developmental disorder of the prenatal period. Some of them are of a genetic, some of an accidental nature, and it is extremely important for the physician to arrive at a correct clinical diagnosis because so many other problems are involved, like the question of more children, genetic factors, reasons for the anomalies, and possible prevention.

Dr. D. Denny-Brown: As I understood Dr. Yakovlev's thesis, the primitive cerebral vesicle can develop into the corresponding region of brain, except for one symmetrical segment. This defective segment may remain as a thin-walled cyst, either without any recognizable neural structure in its wall, or with a layer of poorly differentiated cerebral tissue. Such a process would imply the action of some restricted agent on the anlage of cerebral tissue of the affected region of the cerebral vesicle at a very early stage, different from the diffuse effect of radiation, and sharply restricted in area. Did Dr. Yakovlev find any remnants in the cyst wall to give any suggestion of the nature of the noxious agent? Is there always a cyst wall representing the original cerebral mantle? The failure of development of corpus callosum suggests that the remnants of the vesicle were not such as to serve as a medium for the growth of nerve fibers. Do any of the aberrant fibers that entered the fornix system follow it down to the hypothalamus and cross there?

Dr. Bertram Selverstone: I was interested about the hydrodynamic changes which result in a wide open communication between the ventricle system and the subarachnoid space. Would Dr. Yakovlev say a few more words about that?

Dr. Yakovlev: In regard to Dr. Wegner's question I would say that there is definitely a great difference in vulnerability of the brain, depending on the stage of its development. Generally it seems that embryos up to three months are better protected against hazards such as asphyxia, the ultimate cause of damage to all living tissues, especially the brain, than are fetuses in latter stages of gestation. Secondly the effect of any noxious factor will differ according to whether the factor comes into play before or after the organogenesis of the neuraxis is completed, which is about the end of the third month. The factors operating during the first two months will generally tend to cause greater deviations from normal in the ultimate form and structure of the neuraxis than those that come into play later during the fetal period.

In regard to Dr. Sweet's question. The radiation effect on living tissues not only interferes with the morphokinetic processes of the organogenesis of the developing brain, but also and unavoidably kills the tissues, producing severe destructive lesions. Dr. Hick's work throws much light on the *causal origin* of malformations resulting from the effects of radiation. A yet they do not permit us to learn much about the *formal origin* of the monstrosities of configuration and structure of the nervous system. There are then two distinct problems in the prenatal, embryonal and early fetal pathology: one problem is what does the damage—the causal origin. The other problem—at what time of development the malformation arises and with what consequences to subsequent development of structure and function of the brain. This is the problem of origin of these peculiar brains which I have shown you tonight. The material coming from the autopsy rooms usually affords very little if any data to answer the question of what caused them. On the other hand a careful anatomical study of these specimens in serial sections, which is "sine qua non" in this matter, helps us to recognize in the departures from the normal the basic principles which govern the structuring and organization of the brain as an organ; they reveal the formal origin of these defects. The nature of the material with which I deal is not suitable to answer the question of causality in the narrow sense of etiology of these malformations. Only experiments such as for example those of Dr. Hicks will, with further improvement in the methods used, give us insight into the etiological factors which cause these malformations. There is no contradiction between these two methods, i.e., the inductive experimental and the analytical morphological. They are mutually complementary; both are necessary.

Dr. Denny-Brown brought up an important issue. Indeed the isolated ectopic islands of differentiated cerebral substance containing nerve cells, glia and myelinated fiber bundles in the subarachnoid spaces of the pia-ependymal membrane roofing the defect in the cerebral mantle are extremely common. It is impossible to assume that by some mischance they have been split off from the brain itself and pushed into the subarachnoid space to continue their elaborate histogenesis almost undisturbed. It is much more likely that these islands of cerebral substance in subarachnoid space have developed there in situ with the primitive meninges from the elements of the ectoblast before these elements became irrevocably committed to develop

into the nerve cells, glia or soft meningeal envelopes. To my way of thinking these and other similar findings in the schizencephalic malformations of the end brain bespeak a disorder of the regional induction processes occurring as early perhaps as the presomite or early somite stage, i.e., in the 2nd or 3rd week of gestation.

Dr. Selverstone asked about the nature of hydrocephalus in these malformations. The hydrocephalus is clearly not the cause but the function of the arrested development of the cerebral wall. If the defect is limited to a small area of the brain wall while the surrounding parts continue to develop normally there is usually no hydrocephalus. If, on the other hand, the area of arrested development of the cerebral mantle is extensive and, especially, if the defect occurs near or in the roof plate, the thin vesicular brain wall yields passively and adapts to normal pressure of the fluid contained within the ventricle and so the hydrocephalus "ex loco minosis restentiae" results. In the case of these cleft-forming defects in the cerebral mantle there is no evidence, nor a need to explain the hydrocephalic enlargement of the usually single ventricular cavity of these brains on the basis of obstruction of cerebro-spinal fluid circulation or disturbance of its production and absorption.

THE RESPONSE OF THE CENTRAL NERVOUS SYSTEM TO FOCUSED ULTRASOUND—EXPERIMENTAL RESULTS. *Walter C. Cotter, M.D., Walle J. H. Nauta, M.D., Theodor F. Hueter, Ph.D.,* and *H. Thomas Ballantine, Jr., M.D.*

An ultrasonic generator was constructed with two aims in mind: 1) production of focused ultrasound, and 2) great flexibility in the parameters that determine dosage. By means of a lens, ultrasound has been focused into a region measuring 0.9 x 9 mm. at 2.5 mc. and 2.5 x 35 mm. at 1 mc. The focal intensity can be varied from 100 to 2000 watts/cm.[2] The production of pulsed ultrasound with variable pulse period is possible.

That small lesions could be produced with this apparatus was demonstrated when mice were rendered monoplegic by irradiation of one half of the spinal cord. Production of lesions in stereotactically predetermined sites in cat brain showed the accuracy with which a region of focal damage could be predicted.

Investigations into the parameters determining dosage have been started, using as an end point weakness of one or both hind legs of mice irradiated over the tenth thoracic vertebra. We have used pulsed ultrasound at a constant duty cycle of 0.4 sec. pulse width and 1.0 sec. pulse period, but have varied the number of pulses and the intensity of ultrasound. A statistical analysis of data on over 800 mice, irradiated with ultrasound at 1 mcps. and 2.5 mcps. has established a relationship between dosage and biological effect which indicates there is a threshold of ultrasound amplitude below which no damage occurs and that above this threshold damage depends more strongly on the amplitude of the ultrasound used than on the duration of exposure.

Using these relationships, we are presently engaged in producing lesions graded as to size and shape in cat brain. It has been possible, for example, to selectively destroy the ventral anterior nucleus of the thalamus by a proper dose of focused ultrasound. It is hoped that these investigations will reveal the degree of accuracy with which a cerebral lesion of a specific size and a specific shape can be produced in a specific location without undesired side effects. Only with such knowledge will it be possible to decide whether this tool is ready to be taken out of the research laboratory and put into clinical use in neurological surgery.

Discussion

Dr. Thomas Jones: What are the possibilities for further localizing the lesion by having multiple focus or crystal and lens with radiation at 90° angles?

Dr. James C. White: Do they envisage whether this will be a possible method of destroying tumor tissue? If it can be focused accurately enough, it might be a way of dealing with areas where a surgeon cannot use a knife. It will take a lot of future work to focus the area of transection. There will be a great potential for using it for leukotomy for psychiatric patients and patients with pain. It will have to compete with lesions being made by the introduction of radioactive isotopes, or with radio frequency currents that can be placed so accurately. You have seen the work of Lindstrom in destruction of areas 9 and 10 in ultrasonic lobotomy. I have admired our speakers this evening for wanting to learn all about the effects of ultrasound on animal tissues in the laboratory before trying to take it into the clinic. I think Lindstrom has gone at it the other way around.

Dr. Walter Wegner: About this means of destroying tumors, would the authors tell us about the acute reaction of tissues? Is there severe swelling? Would one have to consider that in using it for tumors?

Dr. Raymond D. Adams: I examined two or three of these sections and there are several unusual features. Everyone wants to know what ultrasound does to the elements within the nerve tissue as well as how accurately this agent can be focused on the nervous system. In the sections I saw with a fairly intensive radiation there is destruction of nearly every tissue element; cell, myelin, astrocyte, microcytes and even blood vessels are completely destroyed. There is almost no hemorrhage in the lesion, suggesting that these must be sealed off almost at once by that which damages the tissue. It will be of interest to the neuropathologist to see if grading doses of this will wipe out parts of the tissues more than others.

Dr. Harry C. Solomon: What are long-term effects of ultrasonic irradiation, say three, six, or nine months later? Will it be a continuous picture of destruction? Is it possible to give a dosage that is sufficient to concuss or knock out the activities and then have recovery take place in the course of days thereafter?

Dr. Ballantine: I would like to express my appreciation for the discussion and the questions raised regarding this relatively new technique. As to techniques of multiple focusing, there is a feasible method which is used by Fry and the University of Illnois. It produces a more punctate lesion but the multiple focusing method is more difficult in application. We are not convinced that it is necessary to use a multiple beam technique to get a small area of destruction.

As to the ultrasonic treatment of brain tumors: there has not been any evidence that abnormal cells are more sensitive to ultrasound than normal. However, all cells can be destroyed within a certain region. If such destruction would allow for shrinkage of tumor without damage to vital areas of normal brain, some symptoms might be alleviated.

As to edema: this must be discussed in terms of dosage and how the ultrasound is applied. If the method of application is such as to produce gross heating of tissue, a great deal of edema will result. If the tissue is "concussed," it is unlikely that as much edema will result as if the tissue were burned. Dr. Adams has put his finger on the crucial point—dosage. We do know that both heat and concussive effects are present, and it is most difficult to separate these two effects and to analyze them.

In answer to Dr. Solomon's question as to the long-term effects of ultrasonic irradiation, there is no reason to believe that there is continuing damage following ultrasound as there is in x-ray treatment. Whether brain tissue can be "concussed" in such fashion as to give a temporary syndrome that can be evaluated we do not know. There are reports that central nervous function has been interrupted for a period of time, following which recovery has taken place. This we have not seen, however.

BOOK REVIEWS

Books received are acknowledged and given a brief description in this section. Selections serving a variety of interests will be made from these for a more extensive review, as far as space will allow.

Perry, John Weir, THE SELF IN PSYCHOTIC PROCESS. [Berkeley and Los Angeles: University of California Press, Pp. xvii + 184. $5.00.]

This book is essentially an extraordinarily comprehensive case history of a patient diagnosed as a catatonic schizophrenia with paranoid trends. The author is a Jungian analyst and has presented his material as a discussion of the emergence of the "self" as manifested through the content of the patient's productions.

A case synopsis and a complete case history of the standard type are included with emphasis on the delusional material. Initial delusions are first presented and the author then gives a running account of his interviews with principal focus of attention directed towards the emergence of the central archetype, rather than to the therapeutic approach. Drawings correlated with the interviews are discussed and many are reproduced in color. Relationship of these drawings to symbols is next pointed out and interpretation of the psychopathological processes from the viewpoint of the Jungian theory are discussed in detail. This section is filled with material which may be acceptable or not to the reader, depending on his interests and training, but will, either way, be of interest.

The second half of the book is devoted to a discussion of symbolism and much important information concerning the quadrated circle in East and West is evaluated. Finally, the author discusses the psychology of such symbolism from his own experienced viewpoint.

An appendix contains the patient's record and a very extensive section of notes and bibliographical materials. Workers oriented in Jungian attitudes will find the book most useful and worthwhile. Other experts trained in different approaches will find the book difficult to understand since the author's writing style is not the simplest. The studies of symbolism also will require some previous knowledge of the Jungian approach and certainly the reader should have at least read "The Secret of the Golden Flower" before beginning this one.

DOUGLAS M. KELLEY, M.D.

Joseph Wortis, M.D., FRAGMENTS OF AN ANALYSIS WITH FREUD. [New York: Simon & Schuster, 1955. Pp. 208. $3.00.]

The latest and possibly the most fascinating appraisal of psychoanalysis is Dr. Wortis' compact and brilliant report of the four months he spent with Freud in 1934-35. These sessions, undertaken as a didastic analysis, complemented a training program in the study of homosexuality under the sponsorship of Havelock Ellis and Adolf Meyer.

Dr. Wortis' notes of these sessions constitute the main body of the book. From it we obtain many new and lively insights into Freud, his theories not only of psychoanalysis but of other treatment procedures, and his opinions of his contemporaries and colleagues, of the role of men, women and children in society, of biology and anthropology, of political movements and of himself.

Freud no doubt realized that the interviews were being recorded for this was not an uncommon practice among his students. Thus, with the complete context given for each opinion and remark we have, in effect, an intimate picture of him at age 78, after a lifetime of experience, fully aware of all the criticism leveled against him and phychoanalysis, but still determinedly holding his own. For Freud did not waver in his metapsychological beliefs. He insisted that "everything is unconscious," and, "we can't bother with the biologists, we have our own science . . ." It apparently did not matter if discrepancies appeared, for "we

must go our own way." Dr. Wortis believes that "Freud was a person of resolute honesty, motivated not only by an intense scientific curiosity but by a high level of human feeling as well." (On which opinions one may take some issue and suggest that they may well be a projection of the author's qualities instead as they are revealed in this book). Wisely choosing to publish this material after twenty years of experience in psychiatry, Dr. Wortis in the final section is enabled to present an evaluation of Freud and Freudianism that is underscored with the healthy perspectives of a mature scholar; for in the interim he has made his own noteworthy contributions to pediatric psychiatry, physiological treatment procedures and the social factors in psychopathology.

It is thus anticipated that the book will bring a number of enjoyable and profitable hours to many people. These will include those who have long been uncomfortable in the irrational aura of deification which has enshrouded "The Master" although each reader will no doubt draw personal conclusions, depending on his bias and orientation. At any rate, ample and documented material is offered in sincere, objective terms which at the same time provide an interesting glimpse into the professional and personal development of a scientific worker, the author himself.

LEONARD CAMMER, M.D.

BOOKS RECEIVED FOR REVIEW

ARIETI, S. *Interpretation of Schizophrenia.* Robert Brunner Publishers, New York, N. Y.

FARIS, R. E. L. *Social Disorganization.* The Ronald Press Company, New York, N. Y.

GARMA, A. *Genesis Psicosomatica y Tratamiento de las Ulceras Gastricas y Duodenales.* Remitente: R. K., Buenos Aires, Argentina.

HOFFER, W. *Psychoanalysis—Practical and Research Aspects.* The Williams & Wilkins Company, Baltimore, Md.

KRABBE, K. H. *Opuscula Neurologica.* Ejnar Munksgaard, Copenhagen, Denmark.

MACDONALD, M. (Ed.) *Philosophy and Analysis.* Philosophical Library, Incorporated, New York, N. Y.

MARTIN, G. J. *Ion Exchange and Absorption Agents in Medicine.* Little, Brown and Company, Boston, Mass.

MAYER-GROSS, W., SLATER, E., AND ROTH, M. *Clinical Psychiatry.* The Williams & Wilkins Company, Baltimore, Md.

MONRAD-KROHN, G. H. *Clinical Examination of the Nervous System,* 10th ed. Paul H. Hoeber, Inc., New York, N. Y.

MORIN, GEORGES. *Physiologie du Système Nerveux Central.* Mason et Cie, Editeurs, Paris, France.

NEWTON, N. *Maternal Emotions.* Paul H. Hoeber, Inc., New York, N. Y.

SCHNEIDER, K. *Psychiatrie Heute.* George Thieme Verlag, Stuttgart, Germany

WILSON, S. A. K. (author); BRUCE, A. N. (Ed.) *Neurology,* Volumes I, II, and III. The Williams & Wilkins Company, Baltimore, Md.

NOTES AND NEWS

GENERAL PALSY MEETING

The American Academy for Cerebral Palsy will hold its annual meeting in Memphis, Tennessee, October 10, 11 and 12, 1955. The sessions are open to members of the medical and allied professions who are interested in cerebral palsy. The three-day program will include instructional courses in the various phases of cerebral palsy as well as a formal program. Those wishing to attend may obtain an invitation upon request from Dr. Robert A. Knight, Secretary-Treasurer, 869 Madison Avenue, Memphis 3, Tennessee. Hotel reservations are to be made directly with Mr. Scott Stewart, Manager of the Claridge Hotel, Memphis, convention headquarters. Dr. Lenox D. Baker, Duke Hospital, Durham, North Carolina, is president of the organization.

YALE UNIVERSITY SCHOOL OF ALCOHOL STUDIES

The Summer School of Alcohol Studies of the Laboratory of Applied Physiology, Yale University, will hold its thirteenth annual session in 1955. The regular session will be held during the four-week period June 27 to July 22 inclusive.

Since 1943, when the first session of the School was held, 1,837 students from 47 states and the District of Columbia, from 9 Canadian provinces and from 17 other countries have attended. The School is organized to meet the needs of a number of categories of professional and nonprofessional people. Address all communications concerning the School to: Registrar, Summer School of Alcohol Studies, Yale University, 52 Hillhouse Avenue, Yale Station, New Haven, Connecticut.

AMERICAN EEG SOCIETY MEETING

The ninth annual meeting of the American Electroencephalographic Society will take place at the Palmer House, Chicago, Illinois on June 10, 11 and 12, immediately preceeding the annual meeting of the American Neurological Association. The scientific sessions will be held on Friday afternoon, June 10, 2:00–4:15 P.M., Friday evening from 8:00–10:15 P.M., and Saturday, June 11, from 2:00–5:00 P.M.

In addition, there will be two Symposia: A Symposium on Micro-Electrodes will be held on Saturday, June 11, from 9:30–12:00 noon under the Chairmanship of Dr. Herbert H. Jasper. The second Symposium on Clinical EEG Interpretation (Head Injuries) will be held Sunday morning, June 12, from 9:30 A.M. until 12:00 noon, under the Chairmanship of Dr. Frederick A. Gibbs.

IV SYMPOSIUM NEURORADIOLOGICUM

The meeting will be held in London from September 13th–17, 1955. Registration will take place on September 12 at the University of London, Malet Street, W.C.1., where the scientific meetings will be held. Radiologists, neurologists and neurosurgeons particularly are welcome.

Subjects for discussion include: i) cerebrovascular disease; ii) the localization of intracranial tumors by means of isotopes; iii) applications of stereotaxometry; iv) new techniques; and v) various proferred papers on a variety of neuroradiological subjects. Scientific and technical exhibitions are being arranged.

It is hoped to arrange a joint half-day session with the 2nd International Congress of Neuropathology which is being held simultaneously in London. A full social programme has been arranged including visits to places of interest for wives and families of members.

Application forms and further information may be obtained from Dr. R. D. Hoare, Secretary. Symposium Neuroradiologicum, The National Hospital for Nervous Diseases, Queen Square, London, W.C.1.

CHILD RESEARCH CLINIC

The Board of Trustees and Staff of The Woods Schools, in collaboration with The Department of Special Education, Teachers College, Columbia University, and The School of Education, The City College, New York,

held the 1955 Spring Conference of the Child Research Clinic in New York City, May 6 and 7, 1955, on the topic: "The Exceptional Child Faces Adulthood."

CERTIFIED BY THE AMERICAN BOARD OF PSYCHIATRY AND NEUROLOGY, INC.

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